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## Original Articles.

## FURTHER OBSERVATIONS ON ALCOHOLIC PARALYSIS.


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SINCE my last communication[1] the subject of Alcoholic Paralysis has received a great deal of attention from different observers. In this country Hadden[2] has described two fatal cases, in one of which a complete autopsy was made:—in France a thesis by Oettinger[3] has been published, treating fully of the history, symptoms and pathological anatomy of alcoholic paralysis; and another thesis, treating more of those cases which simulate locomotor ataxy, is about to be published. In Germany several papers on this subject have appeared. The more important are by Löwenfeld,[4] Strümpell,[5] Knicke,[6] Lilienfeld,[7] and Schulz,[8] who gives an almost full analysis of the hitherto published fatal cases. Of American neurologists, Hun[9] has contributed one paper containing the report of two cases, one of which ended fatally.

[1] 'BRAIN,' July, 1884. [2] 'Path. Trans.,' 1885, p. 49.
[3] 'Étude sur les paralysies alcooliques,' Paris, 1885.
[4] 'Arch. f. Psychiatrie,' xv. p. 438.
[5] 'Berl. Klin. Wochenschr.,' 1885, No. 32.
[6] 'Deutsch. Medicinal-Zeitung,' 1884, No. 72.
[7] 'Berl. Klin. Wochenschr.' No. 45, 1885.
[8] 'Neurol. Centralb.' 1885, Nos. 19, 20 and 21.
[9] 'American Journal of Med. Science,' April, 1885.

Within the last twelve months I have seen quite an unusually large number of cases of alcoholic paralysis, and several others have come under the notice of my colleagues at the Manchester Infirmary. Considering the widespread attention which this subject has attracted, it may not be out of place to briefly relate some of these cases, and to touch upon some points relating to the history, symptomatology, and prognosis of alcoholic paralysis.

Concerning the history of alcoholic paralysis I find that Magnus Huss[1] has been credited with having given the first description of a paralytic form of chronic alcoholism. On looking up the literature of the subject, I found, however, that an American writer, Dr. James Jackson, had as early as 1822–3 given a very succinct and at the same time very accurate and complete account of alcoholic paralysis.[2] The affection itself, the author names from the most prominent symptom arthrodynia; he states that he chiefly observed the disease among females, and that it is produced by "ardent spirits."

Dr. Jackson not only describes the pain, the paralysis and atrophy, but also gives an account of the "glossy skin"—a trophic change well marked in some of the cases recently seen by myself, and which has also been observed by others.[3] Dr. Jackson's description of the disease is worth quoting, especially as the original is not very accessible. "This arthrodynia comes on gradually. It commences with pain in the lower limbs, but especially in the feet; and afterwards extends to the hands and arms. The hands may be affected first in some instances; and in all cases in an advanced state, the pain is more severe in the feet and hands, than in the upper parts of the limbs. The pain is excruciating, but varies in degree at different times. It is accompanied by a distressing feeling of *numbness*. After the disease has continued a short time, there take place some *contractions* of the *fingers and toes and inability to use these parts freely*. At length the hands and

[1] 'Alcoholismus chronicus,' 1852.

[2] The paper is contained in the 'New England Journal of Medicine and Surgery;' Boston, vol. xi. (third series: vol. i. No. 4,) 1822, p. 351 "On a peculiar Disease resulting from the use of Ardent Spirits."

[3] Oettinger, p. 37.

feet become nearly useless, the *flexor muscles manifesting, as* in other diseases, *greater power than the extensors.* The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The *diminution is especially observable in the feet and hands;* and at some time *the skin of these parts acquires a peculiar appearance.* The same appearance is noticed, in a slighter degree, in the skin of other parts. This appearance consists in a *great smoothness* and *shining*, with a sort of fineness of the skin. The integuments look as if tight and stretched without rugæ or wrinkles; somewhat as when the subjacent parts are swollen, but the skin is not discoloured. Yet in this disease there is not any effusion under the skin, and the character which this assumes, arises from some change in the organ itself." . . . (The *italics* are my own.)

The pains, present in the pseudo-ataxic form, have likewise been noticed by this accurate observer, for he says, "The most characteristic symptoms are manifested in the limbs; but the pain is not limited to these, and other symptoms are exhibited in other parts. The pain sometimes *shoots suddenly up one or both legs*, and in one case it frequently passed up the back and then forward to the pit of the stomach, taking the course of the diaphragm."

According to the more prominent symptoms, I have divided clinically this peculiar affection into two groups: the alcoholic ataxia, and the alcoholic paralysis. This classification has been adopted also by several authors—Löwenfeld, Moeli, Schulz, Strümpell and Krücke—who speaks of it as "Pseudo-tabes of alcoholics."

In considering the ataxic form, we have to distinguish between those cases where there is marked inco-ordination without much paralysis, and those where the gait resembles the ataxic gait, but is in some measure due to the paralysis of muscles of the lower extremity. The ataxic form represents a milder type; pathologically, however, as is evident from the cases reported by Déjérine,[1] Moeli,[2] and the case given below it is the same affection, namely, a peripheric neuritis.

[1] 'Arch. de Phys. norm. et path., 1884. No. 2.

[2] 'Charité Annalen,' 1884.

Of the purely ataxic form I have observed three cases, two of which have quite recovered, whilst the third died from uræmia, due to a contracted, gouty kidney.

The *first* case was a man, æt. 32, whom I saw with Mr. Fitzmaurice, of Bury. The patient had been for some years a heavy drinker, and had also indulged, to an extraordinary and almost morbid degree, in venereal excesses, but has never had syphilis. He complained of severe, lancinating and shooting pains in the lower extremities, sometimes in the upper extremities; both lower extremities showed some spots of anæsthesia and of retarded sensibility; the muscles of the calf were painful on pressure; there was no atrophy or paralysis of either upper or lower extremity, but there was marked inco-ordination. With his eyes open the patient could walk fairly well, and lifted the feet well from the ground, the heels coming down first; with his eyes shut, he showed marked ataxia; there was also distinct inco-ordination for finer movements in the upper extremities; the tendon reflexes were absent; no Argyll-Robertson symptom; no ophthalmoscopic changes. The patient gave up the use of alcohol, and completely recovered; and Mr. Fitzmaurice informs me that the tendon-reflexes, which remained absent for some time after the recovery seemed complete, have now returned.

The *second* case is a patient who is a boarder at the Cheadle Asylum, suffering from Dipsomania, and whom, in my capacity as one of the visiting physicians to that Institution, I had an opportunity of seeing frequently. For the notes, I am indebted to Mr. Reynolds, M.B., one of the assistant medical officers.

The patient, æt. 33, single, had an attack of sunstroke at Ceylon when 26 years old, and after that became very nervous and took to alcohol. He came to England, stayed some time, and drank very heavily; went back to Ceylon, and finally returned in 1882. He went to a Home for Inebriates, then to Loxley Hall, and finally came to Cheadle. He has never had syphilis.

His brothers are heavy drinkers, and one of them suffers from epileptic mania. In May 1884, he commenced to complain of pains in both legs and feet, especially on the dorsal surface of the feet near the toes, which were swollen; the pains were

shooting in character. There was hyperæsthesia of the skin over these parts, and some spots of anæsthesia; he had also pains in his fingers. He became very irritable in temper, lost his memory for recent events, and refused his food. There was loss of tendon-reflex, some slight atrophy of both legs, and marked ataxic gait. There were no bladder or rectum symptoms; the atrophy increased, the pains, however, became less, and the patient walked better. Towards the end of September 1884, the pains ceased altogether; the atrophy, however, remained, yet the patient walked much better. At the beginning of March 1885, he had pains over the distribution of the left supra-orbital nerve; he again complained of pains in his legs, burning sensations in the soles of his feet, and cramp in his legs.

In May 1885, I saw the patient myself. I found him somewhat irritable; otherwise but little affected mentally, except that he had lost his memory for recent events.

The gait was feeble and slow; he walked with the help of one stick, but his gait was distinctly ataxic, and became more so when he attempted to walk with his eyes shut. There was some emaciation of the lower extremities, but no marked paralysis of the muscles; the movements of the extensors of the toes were, however, sluggish. There was some slight atrophy of the muscles of the back and also of the arms, but the patient could flex and extend both fingers and wrists very well.

The patient complained of shooting-pains down the legs to the toes, occurring in paroxysms, and leaving a numb feeling behind. Firm pressure on the soles of the feet and on the legs was very painful. The patient also noticed a cold feeling when touched by any object. (This symptom Mr. Reynolds had noticed on several occasions previously.)

The limbs showed no vaso-motor disturbances.

The tendon-reflexes were absent.

The galvanic reactions were taken by Mr. Reynolds.

The contractions were slow, and followed by fibrillar tremors.

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| Extensors of the foot . . | ACC and AOC, with 10 cells.<br>KCC, with 15 cells. |

A very strong magneto-electric current was necessary to get contractions of the extensors of the toes and calf-muscles.

The muscles of the upper extremities reacted well.

There were no eye or bladder symptoms.

There was loss of appetite, craving for drink; the tongue was furred; there was morning vomiting and occasional hæmatemesis.

The bowels somewhat constipated. The liver and spleen were not found to be enlarged.

The pulse was feeble and irregular.

As the patient refused to eat unless he had some drink, he was allowed beer. For the pains he had morphia injections at night, which gave him great relief.

The patient soon began to improve, and again went to Loxley Hall. He has returned from there recently, and is now quite well. He has gained flesh, he has no pains, there is no ataxia, and he shows no abnormality whatever in walking. The tendon-reflexes are normal, and the patient is now only kept as a boarder at his own request. He has never discontinued to drink beer, but avoids all other stimulants.

The *third* case was a female patient, æt. 53, who was admitted into the Infirmary, complaining of pains of shooting character in her lower extremities. Three years ago she suffered from gout in her hands. For some time she has been troubled with paroxysms of pain in her legs, without any swelling of the toes or any of the joints; she has also noticed that her gait was awkward, and that she could not walk well in the dark. On admission, it was found that she had a typical alcoholic appearance; the skin was dry, there was a nodular swelling on the metacarpo-phalangeal joint of the left index-finger. The patient looked thin; there was no œdema, and there was no marked paralysis of any of the muscles. She could walk, but felt weak on her legs; her walk was ataxic, and it was impossible for her to stand or walk with her eyes shut. She could move her limbs freely when in bed; the tendon-reflexes were absent; there was some anæsthesia of the skin of the lower extremities; but very marked hyperæsthesia of the muscles of the calf and of the muscles of the forearm; pressure on these parts caused most excruciating pain. The heart was found hypertrophied; the urine of sp. gr. 1010, profuse in quantity, contained albumen and granular casts; there was anorexia with

vomiting. The pupils reacted well, the fundus of the eye was normal.

Some time after admission the patient became delirious; there was incessant vomiting for twelve hours, and then the patient had a convulsion and died.

At the post-mortem examination made by Dr Harris, Pathologist to the Infirmary, the kidneys were found small and granular; the left weighed 1½ oz., the right 1 oz.; on section, the cortical substance was found very much diminished, and streaks of urate of sodium were seen passing to the medullary part. Microscopically, marked interstitial nephritis was seen, with extensive deposits of urate of sodium crystals in the renal tubes. The heart was very much hypertrophied, and weighed 13½ oz.; the myocardium was healthy, the valves normal. The liver weighed 3 lbs., and microscopic examination showed marked amyloid changes, together with monolobular cirrhosis, the fibrous tissue being still of very embryonic type; the spleen weighed 11 oz. The brain was anæmic; the ventricles contained more fluid than usual; the membranes of the brain were healthy; the pia mater, however, slightly opaque. The pons, medulla, and spinal cord had a perfectly healthy appearance, and were of firm consistence. The muscles of the leg and forearm were thin and pale. The sciatic, the ant. crural, and the musculo-spiral nerves were preserved for microscopic examination, along with the cord, and some muscles from the back of the leg.

The spinal cord, examined carefully after having been hardened, was found perfectly normal in all its parts.

The sciatic nerve appeared thin, grayish, and was surrounded by a great deal of adipose tissue. Vertical sections showed, when treated with perosmic acid, and stained afterwards with picrocarmin, a moniliform appearance of the nerve tubes, due to breaking up of the myelin; the nuclei were increased, and there was also some interstitial cell infiltration. Transverse sections showed in some few places an increase in diameter of the axis-cylinder, and again the interstitial infiltration.

The muscles showed chiefly increase of the muscle nuclei and an interstitial deposit of small round cells, and in some few places the striation was not well marked.

This case, both in its clinical history and in the post-mortem appearances, resembles very closely the first of the two cases described by Déjerine[1] as peripheric nerve-tabes, and there can be no doubt that both the cases described by this author are cases of alcoholic ataxia. We omitted in our case to remove portions of the skin, and I regret very much not to be able to state anything about the condition of the subcutaneous nerves, which were found markedly affected in Déjérine's cases.

The following case, which was admitted into the Infirmary only a few days ago, shows the combination of alcoholic ataxia with alcoholic paralysis.

W. B., æt. 41, was admitted into the Infirmary Nov. 10, 1884. The patient had been very intemperate and has a distinctly alcoholic appearance. He has had rheumatic fever, and has lately been very much troubled with pains in his limbs. He stated that he has had three attacks like the present, but not so bad, from which he recovered, after rest and abstention from drink.

The patient looks strong and stout; some of the muscles feel flabby, but there is no marked atrophy, though paralysis of some of the muscles is distinct.

In the upper extremity there is marked paralysis of the extensors of the fingers and of the wrists on both sides; some of the other muscles (triceps, deltoid) are slightly affected; the flexors act very well. Such movements as the patient is able to carry out show some inco-ordination.

In the lower extremity the extensors of the toes and of the big toe are but slightly affected; the peronei on both sides, however, are considerably paralysed; the arch of the foot is flattened, and the inner border is raised, whilst abduction is impossible.

The patient is able to walk with some assistance, but his walk is ataxic; he keeps his legs apart, and has to look to the ground for fear of falling; he cannot walk with his eyes shut. Isolated movements with either of the lower extremities show equally marked inco-ordination. With the eyes shut the patient does not know exactly the position into which his limb is put.

[1] 'Arch. de Phys.,' 1884, p. 231.

The sensory phenomena are those of alcoholic paralysis. The patient complains of shooting pains in his legs. There is cutaneous anæsthesia in both upper and lower extremity, irregularly distributed, with extreme muscular hyperæsthesia. There is also great pain if the skin apart from the muscles is firmly grasped. The sense for temperature is affected in both legs; the patient rightly distinguishes a cold body, but contact with a hot body gives him a sensation which he compares to that of an electric shock. There is some analgesia, and the prick of a pin is felt only after some time.

The tendon-reflexes are absent; the superficial reflexes fairly normal.

The pupils are normal, and react to light and accommodation; the special sense-organs are normal.

The mental condition of the patient shows symptoms which are often found in cases of alcoholic paralysis, and which have been noticed by other observers, without, however, laying great stress on them. The patient answers questions rationally; his memory, however, is very defective, and he suffers from delusions. Thus he tells you that he gets up every day, goes into the next ward, and converses with the other patients, though, as a fact, he has never left the ward since his admission. When further pressed, he even gives a description of the ward, details his conversation with the patients with a minuteness and readiness which is astonishing. I have noticed exactly the same peculiarity of the mental state in some of the other cases to be presently described.

The condition of the other organs calls for no special notice. The heart-sounds are normal, but weak; the pulse 120, compressible; appetite fairly good; the liver is normal; urine free from albumen.

We have so far been able to take the electric reactions to the galvanic currents only once, and muscles showed degenerative changes.

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| Peronei of right side . . | KCC, with 30 cells. |
| | ACC „ 25 cells. |

The other cases which I have as yet to describe belong to the typical class of alcoholic paralysis, characterised by

well-marked paralysis with atrophy, affecting chiefly the extensors of the fingers and toes.

The paralysis and atrophy sometimes come on very acutely, at others more slowly. When these cases come under observation, the patients are as a rule unable to stand or walk, and it is therefore not easy to make out whether the paralytic stage is here preceded by an ataxic stage. As the sensory phenomena in these cases are the same as in the first group, it is highly probable that where the paralysis comes on slowly, pseudo-ataxic symptoms, as in the case just given, precede it. Of this group I have within the last twelve months seen four cases, all females, two of which have quite recovered, one is beginning to improve, whilst the fourth, when last heard of, had as yet shown no signs of improvement. As the symptomatology of these cases is now sufficiently well known, I will very briefly relate the cases, only drawing attention to particular points.

Case I.—A lady, æt. 25, whom I saw with Mr. Renshaw, of Altrincham, in August 1884.

The patient had freely partaken of spirits, and had a constant craving for stimulants.

She had complained of some pain for some time; the pains were soon followed by paralysis and atrophy of the extensors of both fingers and toes, with paresis of the other muscles, so that the patient could neither stand nor walk, and could raise herself only with difficulty from the recumbent posture. Cutaneous anæsthesia and muscular hyperæsthesia were well marked. Tendon-reflexes absent, superficial reflexes much diminished.

The patient exhibited the same peculiar delusions just referred to. When I saw her she told me that she could walk very well, that she had paid several visits that morning, and minutely described all she had seen and done when visiting her friends. When her helpless condition was pointed out to her, she became highly emotional, and burst into tears.

The patient was removed to a small cottage, and attended by a reliable nurse, so that she had no access to drink; she completely recovered in three months. Mr. Renshaw informs

me that she now walks very well, and that the tendon-reflexes have reappeared.

Case II.—A lady, æt. 42, whom I saw with Mr. Kitchen in September, 1884. Her symptoms resembled those of Case I. The paralysis had come on rather more slowly, and the atrophy was much more marked, and affected the greater part of the body. This patient also had the same peculiar delusions. Though entirely confined to bed, she told us that she went out every afternoon for a walk to the seaside; that she sits on the sand at the seashore, watching the waves and the passing steamers. (Her residence was at least twenty miles away from the sea.)

It was astonishing to listen to the account she gave of her seaside rambles, which had every semblance of truth, and yet was entirely imaginary.

We advised the patient's removal, if possible, as we were sure that the patient, unknown to her husband, was still supplied with stimulants. Our suggestion was, however, not acted upon, and when last I heard of the patient, she was still in the same helpless state, and rather more emaciated.[1]

Case III.—E. C., æt. 33, seamstress, married, was admitted into the Infirmary, Dec. 3, 1884, suffering from typical alcoholic paralysis.

She had suffered from "rheumatic" pains at different times. Three months before admission, the pains in her arms and leg became much worse; her legs often "gave way" when she attempted to walk, and a corresponding weakness was noticed in her arms. The weakness increased to such an extent, that she was obliged to keep to her bed, and had to be fed, not being able to use her hands. She admitted having partaken of alcohol in large quantities.

The patient had a characteristic alcoholic appearance. The muscles of the upper and lower extremity felt flabby; the extensors of the fingers and toes were quite paralysed; the

[1] Since the above was written, I have heard from Mr. Kitchen that the patient has lately much improved, and is now able to walk with the help of sticks. She has, however, the same delusions still. She has entirely abstained from spirits, but takes several glasses of beer every day.

hands presented the characteristic drop-wrist; the feet, the paralytic club-foot; the flexor muscles of the fingers and toes were paretic; the arm and shoulder muscles were fairly strong. The tendon-reflexes were absent, the superficial reflexes normal. There was also cutaneous anæsthesia in the legs and forearms, with marked muscular hyperæsthesia on firm pressure. The sense of temperature was affected on the outer side of each leg; the patient was able to distinguish cold, but not heat. The paralysed muscles were atrophied to a much greater extent than the paretic muscles.

The patient complained of loss of memory, but otherwise her mental condition was normal. The right pupil was irregular from an anterior synechia.

The application of the electric current was very painful.

A very strong faradic current produced no contraction in the extensors of the hand; the flexors responded to a strong current, but not to a weak current. With the galvanic current directly applied to the muscles, there was found to be degenerative reaction in the extensors of the fingers, the extensor carpi ulnaris, the extensor communis digitorum, and extensor longus pollicis. Some of the flexor muscles also showed degenerative reaction.

The patient remained in the hospital till March 1885, when she left us somewhat improved; she has since then completely recovered. She has lately presented herself in the out-patients' department of the Infirmary, and no trace of any paralysis or atrophy could be discerned. The tendon-reflexes had reappeared.

Case IV.—M. E., æt. 43, was admitted into the Infirmary on July 13, 1885, under Dr. Leech, who kindly transferred her to me. The patient admitted having partaken of stimulants (whiskey and gin), and gave the usual history of rheumatic pains, with which she had been troubled for some time, followed by weakness which had increased to such an extent that she was rendered quite helpless. On admission, her condition resembled very much that of Case III., except that the paralysis was much more extensive. The patient was very stout; the muscles flabby; she was unable to raise herself from the recum-

bent posture; hands and feet were flexed; there was marked wrist-drop, the elbows were bent, the knees drawn up. The shoulders and hips could be moved, but the movements were sluggish, and when passive movement was attempted, the patient screamed with pain. The tendon-reflexes were absent, the superficial reflexes not well marked.

There was again cutaneous anæsthesia for tactile impressions, with well-marked muscular hyperæsthesia, affecting upper and lower extremities, and to some extent also the muscles of the abdomen. The sense for temperature was normal.

The skin on the dorsal surface of the fingers of both hands had a marked glossy appearance.

The patient's mental condition is affected; she answers questions rationally, and shows no incoherency in her answers, but she has delusions. She fancies she is at home, &c.; she is very restless at nights, and sleeps but little; she is also very emotional.

The tongue is covered with thick white fur; there is anorexia, occasional vomiting, and constipation. The liver and spleen are normal in size.

To the faradic current some of the muscles (triceps and extensors of the fingers and toes) do not respond at all; some, *e.g.* the biceps, supinator, the interossei, contract with a current of medium stregth.

The application of the electric current is so painful, that the galvanic reaction could only be tried in the most affected muscles, and here marked degenerative reaction was found, thus:—

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| Extensors of fingers (left) . . | ACC = 15 cells. |
| | KCC = 25 „ |
| | AOC 20 „ |

The flexors also showed degenerative reaction, thus:—

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|---|---|
| Flexors of fingers (left) . . | ACC = 30 cells. |
| | KCC = 40 „ |
| | AOC = 45 „ |

The patient has, up to now, made some slight progress; the extensors of the fingers on the left side show some power, likewise the extensors of the toes on both sides. There are

now, however, very marked contractures of the elbows, shoulders and knees, with absence of deep reflexes. The contractures are more pronounced on the right than on the left side. There is also some slight swelling over the back of the right hand (the tenosynovitis of Gubler, as in lead palsy).

The muscular hyperæsthesia is better.

The mental condition of the patient has undergone marked improvement; she has now no more delusions, and has no recollection of her former delusions.

I have no wish to prolong this paper by any further remarks. The symptoms of the disease are so clear, that there is no difficulty in diagnosing the affection.

The peculiar form of delirium seems almost as characteristic, though by no means as constant, as the muscular hyperæsthesia. Glynn[1] has already drawn attention to it. It occurs in chronic alcoholism without paralysis; and Mr. Mould, Superintendent of the Cheadle Asylum, informs me that he has constantly met with it amongst dipsomaniacs.

As regards the pathology, we have now abundant evidence in the post-mortem appearances that alcoholic paralysis is a multiple peripheric neuritis. This view is again supported by the third case of alcoholic ataxia given above. For an analysis of the results of the post-mortem examinations in alcoholic paralysis, I must refer to Schulz.[2]

[1] 'Liverpool Medico-Chirurgical Journal,' July, 1883, p. 374.

[2] 'Neurol. Centralb.,' 1885, No. 21.

# ON A CASE OF MULTIPLE SPINAL AND CEREBRAL TUMOURS (SARCOMATA), WITH A CONTRIBUTION TO THE PATHOLOGY OF SYRINGOMYELIA.

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THE following case came under my observation a few years ago when resident in the Manchester Royal Infirmary; and as I was able to watch the course of the disease for a period of eight months, I have considered it worth while to now put it on record, since it possesses many features, not only of clinical, but of pathological interest.

The patient was under the care of Dr. Morgan during the greater part of his stay in the Infirmary, and it is to him that I am indebted for his kind permission to publish it.

W. D., a married man, aged 38, was admitted at the end of July, 1881, to the Manchester Royal Infirmary, under the care of Dr. William Roberts, complaining of weakness of the left lower extremity with aching pains in both legs. He stated that up to the commencement of the present attack he had never had any serious illness. No history could be obtained of any syphilitic taint.

His illness commenced about eighteen months previously, with aching pains in the lower extremities; but these were more marked on the left than the right side; in the course of a few months the left leg became weaker than the right, and this soon was so marked that, according to his own description, "the left leg would occasionally slip from under him, and he would fall to the ground." It was not until about nine months since that he noticed the wasting of the muscles on the front of the left thigh, which at the time of his admission was such a marked feature. At no time had he

noticed any numbness of either of the lower extremities, neither has he had any bladder troubles.

On August 1st, 1881, we have the following note as to his "present state":—

Patient is 5 ft. 4 in. in height, of healthy but not robust appearance; he complains of aching pains in both lower extremities, but they are much worse in the left than the right leg, and are sometimes so severe as to necessitate a sleeping draught being administered; he also complains of weakness of the left lower extremity. He is able to walk short distances, but this causes fatigue; he walks with no special kind of gait, but slight dragging of the left leg is observed. On examination, the chief feature which strikes one is the excessive atrophy of the extensor muscles on the front of the left thigh; the other muscles of this extremity appear also wasted, but not to that extreme degree which the above extensors show. He does not complain of weakness of the right leg, but all the muscles of it appear more or less atrophied. Patellar tendon-reflex of the left leg almost abolished; that of the right present, possibly in excess. At this date we have no note of any cutaneous sensory disturbance. There are no bladder troubles.

On August 6th he was sent to the Convalescent Hospital at Cheadle, but there became much worse, first noticing that the right lower extremity became numb and gradually weaker; it also became a seat of aching pains, which were far more severe than they had ever been in the left leg. He had also difficulty in passing his water, having to strain to do so.

On September 26th he was returned to the Manchester Infirmary, this time under the care of Dr. Morgan.

On his return he was just able to walk, but required great assistance to do so. He took to his bed, to which he was ever afterwards confined.

On October 1st we have the following note:—He is now not only unable to walk, but to sit up in bed, being unable also to raise himself from the horizontal to the sitting posture.

He complains of great pains in the right leg; this is not of a shooting but of a continuously aching character. There appears to be no hyperæsthesia of the skin, but when the

muscles of the leg or thigh are grasped, it gives him great pain. There is considerable wasting of the muscles of both legs and thigh, but that of the left side is more marked than the right, there being very little of the quadriceps extensor of the left thigh remaining. He has now almost complete paraplegia, being unable to flex the thighs on the abdomen, nor can he move the legs at the knee-joints; he can just manage to move the feet at the ankles and the toes on either side.

The quadriceps extensor muscle of the right thigh responds to a weak faradic current, but requires a moderately strong galvanic one to give a response, the kathodal closure contraction occurring with a fewer number of cells than the anodal closure one.

The quadriceps of the left thigh gives no response to the strongest faradic current, but with the galvanic current a contraction is obtained with a powerful current on kathodal closure, but no response can be seen with the strongest current at anodal closure.

The tibialis anticus of either side responded readily to a weak faradic current, that of the right leg to a slightly weaker current than the muscle on the other side. With the galvanic current, the kathodal closure contraction took place with a weaker current than the anodal closure contraction; the kathodal closure contraction occurring with the same strength of current on either side, but the anodal closure contraction ensued with a somewhat weaker current in the case of the right tibialis anticus, than it did with the muscle of the left side.

There is no rigidity of the lower extremities; on the contrary, they are quite flaccid.

Patellar tendon-reflex of the left side almost, but not quite, abolished, that of the right side is now considerably increased.

He has a slight feeling of constriction round the abdomen.

There now exists cutaneous analgesia and anæsthesia over the front and outer side of the lower half of the left thigh; also another small patch of anæsthetic skin, not more than an inch in diameter, exists over the margin of the costal arch

in the right mammary line. No other affection of sensation can be found in any part.

No weakness of the upper extremities.

There is no deformity of the spinal column, and no tenderness is detected on pressure along the spine.

Once since his return from Cheadle the catheter had to be used, on account of retention of urine, but usually he is able to pass his water, though he has to strain to do so. No bedsores, and no joint affections. Temperature normal.

No affection of the circulation or respiration, and the functions of other organs are performed in a healthy manner.

*Nov. 8th.*—He is still unable to move his lower extremities any more than he could a month since, but the continuous pain, and that caused by grasping the muscles of the legs, have both disappeared. He is still unable to raise himself from the lying to the sitting posture. Patellar tendon-reflex of left side almost abolished. That of right side slightly in excess.

The whole of the left lower extremity has now become anæsthetic and analgesic, this extending above to the level of a line drawn through the umbilicus, on the lower part of the abdomen it does not quite extend to the middle line in front. Over the right lower extremity sensation is impaired, though there are no distinct patches of anæsthesia.

The patch of anæsthesia still exists over the costal margin on the right side, and remains much the same in extent as noted a month back.

For the last three weeks he has complained of a heavy sensation over the right frontal region, and about the same time he noticed numbness of the right half of the face.

There now exists marked cutaneous anæsthesia and analgesia over the right orbit. Over the cheek and lower jaw of the same side, there is less acute sensation than on the left side, but there is no complete loss of sensation.

The conjunctiva of the right eye is much injected, and there is a superficial ulceration of the cornea of that eye.

The right masseter and right temporal muscle are considerably atrophied.

There is no affection of taste on either side of the tongue.

No vomiting or vertigo is present.

Patient is very intelligent, and there appears to be no mental disturbance.

No optic neuritis is seen in the left eye; the fundus of the right cannot be seen, on account of the ulceration of the cornea.

No oculo-motor troubles; no difference in size of the pupils of the two sides. No facial paralysis.

Hearing good on both sides, but much better on the left than on the right side. No difficulty in swallowing; no morbid symptoms connected with the circulatory or respiratory systems. Urine free from albumen and sugar.

No stiffness or rigidity of the muscles of the neck.

No paralysis of the muscles of the tongue.

No affection of speech.

He is able to pass his urine more readily, not having to strain so much to do so. Appetite good; bowels constipated. Temperature normal.

No bedsores.

*Nov.* 21*st.*—He is now unable to move either toes or feet at the ankle joints.

Some rigidity of the right lower extremity has come on; while he lies with the left leg fully extended, the right is flexed at the hip and knee joints, and the hamstring muscles stand out in marked relief; we can bring this limb into the extended position, but doing so causes patient considerable pain at the knee.

The atrophy of the muscles of the lower extremities is becoming more marked; this is especially perceptible in the right limb, where the quadriceps extensor cruris has very much diminished in volume, and is now nearly as much wasted as that of the left side, very little muscle remaining on either side; the girth of the thigh, three inches above the upper margin of the patella, is ten inches on either side.

The atrophy of the muscles of the calf and those on the anterior aspect of the leg is now rather more marked on the right than the left side. No patellar tendon-reflex can now be obtained on either side; even on the right side, where it was so much in excess, it is now absent.

No epigastric or cremasteric reflex can be obtained, but the abdominal reflex is present.

The skin on the right lower extremity is almost entirely anæsthetic and analgesic, except that at one or two isolated spots he occasionally recognises the prick of a pin, but he is still able to recognise heat and cold when applied over this limb.

The patch of anæsthesia over the right costal margin is rather more extensive, but it is separated yet by a portion of sensitive integument from the anæsthetic part below.

The anæsthesia over the left lower extremity remains the same; it does not extend higher than the level of the umbilicus, and not quite to the middle line on the lower part of the abdomen.

The anæsthesia over the distribution of the ophthalmic division of the 5th of the right side remains the same; but there is in addition a small patch of anæsthesia immediately below the right eye, while the rest of the skin of the cheek is sensitive, though not so acutely so as on the left side. The cornea has become more hazy. There is no evidence of the involvement of any cranial nerve, except the 5th of the right side.

There is no optic neuritis.

He is very little troubled with headache; there is no vertigo or sickness.

There is now complete retention of urine, and the catheter has to be constantly employed.

For the last ten days there has been a slight evening rise of temperature, while the morning has been normal. This has been constant during that time, but it has not reached a higher point than 100° F.

His general health remains fairly good.

The loss of sensation has now extended until the whole of the right lower extremity as well as the left is anæsthetic and analgesic, though occasionally he can feel the prick of a pin at a few spots over this surface, but these sensitive parts have no regular arrangement.

*Dec. 4th.*—The anæsthesia has now extended, until that over the right costal margin and over the right lower extremity have merged into one. The upper limit of the anæsthesia on the right side is a line drawn through the apex of the xiphoid cartilage, while on the left side it does not extend quite so

high, the upper limit being a point about two inches above the umbilicus.

The atrophy of all the muscles of the lower extremities is most marked, it being much the same on the two sides. There is now observed slight swelling of the left knee-joint, evidently due to effusion of fluid into the interior of the joint; the fluid appears limited to the joint and does not involve the surrounding subcutaneous tissues. There is no redness over the joint and no pain, and patient was unaware of the existence of the swelling. The left ankle-joint appears also a little swollen, but this is less marked than the swelling at the knee.

Retention of urine still exists; there is now also incontinence of fæces.

A small bedsore has appeared in the central line over the upper part of the sacrum.

Anæsthesia exists now over the whole of the right half of the face, except over the lower jaw. The ulceration of the right cornea and injection of the conjunctiva has increased.

The right half of the upper lip is swollen and thickened, and on the inner aspect is an ulcer the size of a sixpenny-piece; it causes him no pain, in fact he is not aware of it, merely noticing the swelling of the lip; opposite this ulcer there is rather a sharp rough tooth.

The palpebral fissure of the right side is smaller than the left. There is no evidence of any other cranial nerves being involved. He has complained lately of a little more pain over the right half of the face and right frontal region.

There appears to be distinct flattening of the left half of the chest below the nipple; the movement here is not so good as on the right side, the ribs appearing to be dragged up, but the chest undergoing very little expansile movement. There is no anæsthesia over this flattened part. Percussion and auscultation of the chest give negative results.

The temperature still shows slight evening rises.

*Dec. 15th.*—The limits of the anæsthesia of the lower half of the body remain stationary; the loss of sensation does not appear to be extending at all. We have a note at this date that the movement of the chest appears much the same on either side; if anything, it is deficient on both sides.

The retention of urine is beginning to give place to incontinence. The incontinence of fæces persists. The urine is acid when drawn off and contains only very little pus.

The temperature has lately shown more considerable variations; for the last three nights it has risen to above 101°; last night it was 101°·6, and the previous evening 102°; it falls to normal in the morning.

There appear to be no local changes in the lungs to account for this pyrexia.

The condition of the right eyeball, as regards the neuroparalytic ophthalmia, remains stationary; the palpebral fissure is still smaller on the right than the left side.

Another ulcer has now appeared, this being on the right half of the lower lip in a corresponding position to the one on the upper lip. The latter ulcer still exists, and neither ulcer extends across the middle line to the left.

No other cranial nerves besides the 5th are involved. There is no optic neuritis. He is now very little troubled with headache.

*Jan.* 10*th*, 1882.—His general condition has changed much for the worse; his appetite fails, and he has lost a great deal of flesh this last fortnight.

There is incontinence of urine, and the fluid is very purulent, alkaline, and offensive.

The temperature chart still shows considerable evening exacerbations.

The condition of the lower half of the body remains the same, except that the effusion into the left knee-joint has disappeared, and the joint has returned to its normal condition.

There is no loss of power or anæsthesia of the upper extremities.

The ulcers on the inner aspect of the lips still persist.

*Jan.* 25*th*.—Patient continues to get worse, he has emaciated very much lately.

The urine is very fœtid and purulent, so the bladder is now washed out frequently with a solution of boracic acid.

There is still a considerable evening rise of temperature; about a week ago he had a shiver, and the thermometer re-

gistered 104·4°, but this temperature was not maintained for many hours, it soon falling to 101·4.

The grasp of the left hand has now become distinctly weaker than the right, but the anæsthesia still maintains the limit it has done for so long, and does not extend.

The expansile movement of both sides of the chest appears deficient; auscultation and percussion of the chest give negative results.

The atrophy of the muscles above the knees appears the same on the two sides, but below the knees it is more marked on the right than left side, a difference which is especially marked in the calf muscles.

During the last few days a red erythematous patch, followed by the development of a bulla, has appeared over the external malleolus of the left leg; a similar formation has ensued on the under and inner aspect of the left heel.

The anæsthesia over the course of distribution of the 5th cranial nerve of the right side still exists, but a great change has ensued in the condition of the right eye. The conjunctival injection has entirely disappeared, and the eyeball appears quite pale, like the left eye; yet the cornea is still opaque.

For the last fortnight a small ulcer, about the size of a split pea, has existed on the skin of the lower eyelid, near the inner canthus, of the right eye.

The ulcers on the mucous surface of the lips have almost healed.

No optic neuritis nor evidence of the involvement of any cranial nerves, except the right 5th.

*Feb. 4th.*—Patient has complained these last few days of rather more headache.

There is still incontinence of urine, but the fluid has become of a clear amber colour, not at all offensive, acid, and contains no albumen.

The temperature is still a little elevated, but does not show such considerable variations as at the date of the last note.

The ulcers at the inner aspect of the lips and at the inner canthus have now healed.

The weakening of the left arm has become very marked,

the grasp of the left hand, as registered by the dynamometer, is 9, while that of the right is 45. Not only is the grasp enfeebled, but the power of movement at any of the joints of this extremity is much lessened. No anæsthesia is found over the arms; the upper limit of the loss of sensation remains the same.

The condition of the chest movement is the same as at the date of the last note, there is very little expansion movement of the lower part on either side.

The bedsore on the sacrum still exists, but does not increase in size.

The bulla over the left external malleolus has given place to a small sore; that under the left heel has dried up.

The skin along the outer side of the left foot has become erythematous, apparently due to pressure, the foot resting on this part. The skin under the right heel has become dark, soft, and rather pulpy, but it is not ulcerated.

*Feb.* 10*th.*—The condition of the left arm remains the same; the triceps tendon-reflex is increased on either side, that of the left distinctly more so than the right.

The chest movements remain the same. Not only below but also at the upper part of the chest there is very little expansile movement, and respiration appears to be chiefly diaphragmatic.

There is effusion into the right knee-joint, which is swollen, but not reddened or painful.

*Feb.* 18*th.*—The effusion into the right knee-joint still persists.

The conjunctiva of the right eye has now again become much injected.

Triceps tendon-reflex is still increased on either side.

*Feb.* 25*th.*—Condition of the left arm remains the same; there is no evidence of the involvement of the right upper limb.

The thoracic movement still remains very defective.

There is still slight effusion into the right knee-joint.

There has now appeared internal strabismus of the right eye, with incapability to turn the eye outwards; the other movements of the right eye and all those of the left are preserved.

There is slight but distinct facial paralysis of the left side.

*March 3rd.*—Patient's general condition is worse, his appetite is not so good; he feels cold and shivers occasionally. The temperature shows considerable variations; last night it was 102·8°, this morning it is 100·6°. The urine has again become very fœtid and purulent.

The left arm has become very rigid, and is firmly applied to the side of the trunk and flexed at the elbow-joint, while the wrist is extended and the fingers slightly flexed on the palm; he is only just able to move this left upper extremity; the triceps tendon-reflex is increased on both sides; but there is no evidence of any loss of power in the right arm.

The condition of the cerebral symptoms remains unchanged, there is no evidence of the involvement of any more cranial nerves; the internal strabismus of the right eye and left facial paralysis persist. Hearing is equally acute on either side.

The conjunctiva of the right eye is now pale, but the corneal opacity exists. The right palpebral fissure is markedly smaller than the left.

*March 11th.*—Patient's general condition is worse; the temperature is more continuously above normal.

The condition of the cerebral symptoms remains the same as last week, except that the left facial paralysis is a little more marked.

While the anæsthesia over the course of distribution of the right 5th still exists, *the sense of taste over the anterior part of the tongue on either side is very acute.*

The upper limit of the anæsthesia, over the lower part of the body, is now about the same on either side, it extending up to a line drawn about 3 inches above the umbilicus.

The left arm remains in the same state; he can only just move it.

The effusion into the right knee-joint still exists.

For the last week he has spoken somewhat indistinctly, but there is no loss of memory for words.

*March 18th.*—He is much worse to-day. His temperature the day before yesterday was 105·6°, and this morning it is 101·8°; with this exception, it has never been below 102° F. for the last six days.

The incontinence of urine and fæces persists. The urine is exceedingly putrid and offensive.

The injection of the conjunctiva of the right eye has now come on again. The movement of the eyeballs is perfect in all directions, except that, through the paralysis of the external rectus of the right eye, he cannot look with that eye to the right.

The facial paralysis of the left side of the face is distinct; it involves the lower facial muscles, the upper facial muscles being quite free; he can close the eye on either side. There is no paralysis of either hypoglossal nerve.

The palpebral fissure of the right eye is still much less than the left; the pupils of the two sides cannot be compared, in consequence of the corneal opacity of the right eye.

*March* 22*nd*.—Patient gradually became weaker, and died last night, not having manifested any other symptoms.

*Post-mortem*.—This examination was made by Mr. A. H. Young, late Pathologist to the Manchester Infirmary.

Body greatly emaciated. Rigor mortis absent. Cutaneous hypostasis was well marked on the ventral aspect of the trunk and extremities (the body has been in the prone position since death, ice being applied to the spine).

A large bedsore existed over the sacrum and a smaller one over the right external malleolus. There was slight greenish discoloration of the anterior abdominal wall.

*Spinal Cord*.—On opening the spinal canal the whole of the nerves of the corda equina were seen to be greatly thickened and to possess almost a cartilaginous firmness. The dura mater was with difficulty separated from the posterior common ligament of the vertebral bodies, *i.e.* in the lower part of the canal.

Five inches above the tip of the conus medullaris, the spinal cord for a distance of 2½ inches presented an area of great firmness, cutting and looking not unlike cartilage; at the periphery small opaque calcareous-looking deposits were observed.

Elsewhere the cord was somewhat softer than normal. In the cervical region there was marked dilatation of the central canal. No other definite naked-eye changes could be distinguished.

*Brain.*—The right side of the pons varolii was almost entirely replaced by a mass of new growth, like that in the spinal cord, of firm cartilaginous consistency. A small nodular outgrowth of this growth penetrated into the substance of the temporo-sphenoidal lobe. Brain-substance in other respects showed no naked-eye changes. The arachnoid membrane was slightly thickened and opaque, and there was considerable subarachnoid œdema.

The Gasserian ganglion on the right side was invaded by new growth, an extension of that of the pons.

*Thorax.*—Old pleuritic adhesions were found at each apex. Numerous subpleural masses (of various sizes) of new formation, flattened in shape, of a whitish colour. Lung-substance generally was normal.

Pericardium showed a single secondary nodule of small size.

Heart, 10 oz., dilated and hypertrophied.

*Abdomen.*—Alimentary canal not examined.

Liver, 4 lbs. 10 oz., congested.

Kidneys, right, 10 oz.; left, 7 oz. Substance of each organ presented numerous small abscesses. Pelvis dilated, and also full of pus.

Spleen, 8 oz., soft and pulpy.

Right knee and hip were opened; they showed hyperæmia of the synovial fringes and some increase of synovial fluid, but in other respects were normal.

*If we briefly summarise* the chief clinical and pathological features of the case, we notice: A man, of about 38 years of age, had suffered for 18 months with aching pains in the lower extremities, more marked in the left than the right, together with considerable weakness of the left leg. After this 18 months he first comes under our observation, complaining only of the above symptoms; we notice considerable wasting of the muscles of the left lower extremity, especially of the extensor muscle on the front of the left thigh; no loss of sensation and no bladder and rectum troubles are yet present. However, in the course of the next month he begins to get much worse; his legs become weaker, and he now, for the first time, notices great weakness of the right leg, which becomes the seat of very severe pains, similar to those observed in the left leg at a

previous date; about the same time he also has to strain to make water. In the course of the next month the weakness so much increases, that he becomes unable to walk, and has to take to his bed, and at the end of this second month he is not only not able to walk, but cannot raise himself from the lying to the sitting posture, and can only just move the toes and bend the feet at the ankle-joint. The lower extremities are quite flaccid and not at all rigid; the muscles are wasted, especially the extensor cruris of the left side. The muscles, however, show no reaction of degeneration.

At the beginning of the third month a patch of anæsthesia appears on the front and outer side of lower half of the left thigh, just above the knee, and another patch of anæsthetic skin is found over the costal arch on the right side in the right mammary line. In the course of the next six weeks the skin over the whole of the left lower extremity becomes anæsthetic up to the level of the umbilicus; in a month more the whole of the right lower extremity has become anæsthetic, and the anæsthetic part below has merged with the patch of anæsthesia over the right costal arch. During this time also the atrophy of the muscles of the legs has increased, and the catheter has had occasionally to be passed to relieve retention of urine.

Soon after the patch of anæsthesia appeared on the left thigh, *i.e.* after he has been under observation about three months, a series of cerebral symptoms begin to manifest themselves: he first notices pain, followed by anæsthesia, over the right eye, and the skin here becomes anæsthetic, and very soon this extends to the skin of the right cheek; ophthalmia also develops on the right side. The motor division of the 5th cranial nerve is also affected. By the end of the fourth month of observation complete retention of urine has ensued.

In the fifth month of observation there is evidence of effusion of fluid into the left knee-joint, without any signs of inflammatory action. In the same month the retention of urine gives place to incontinence, and the urine becomes purulent; at the same time slight pyrexia begins to develop.

In the fifth month, also, a painless ulcer appears on the inner aspect of the right half of the upper lip, and a little later, another on the inner aspect of the lower lip on the same side.

A month afterwards an ulcer appears on the skin of the lower eyelid near the inner canthus.

In the fifth month of observation the palpebral fissure of the right side is smaller than on the left side. Towards the end of the sixth month the left arm begins to become weak and rigid, and the triceps tendon-reflex of that side is increased. In the seventh month this increases, and paralysis of the external rectus of the right eye, and of the facial muscles of the left side, is to be noted.

The urine becomes more purulent and offensive, and the pyrexia greater. Bedsores appear on the sacrum, and bullæ are seen about the heels and malleoli. He gradually becomes weaker, and dies, after being under observation for about eight months, and having suffered from spinal symptoms for a little over two years.

At the autopsy is found a sarcoma, matting together the nerves of the corda equina; another sarcoma exists in the dorsal region, replacing the cord for a distance of two and a half inches, but the shape of the cord being here, nevertheless, still preserved; and a third tumour replaces the right half of the pons, and involves the 5th nerve and the Gasserian ganglion. Marked dilatation of the central canal of the spinal cord.

Secondary tumours in the lungs and pericardium. Suppurative nephritis.

*Microscopical Examination and Commentary.*—The various new growths—viz. that in the corda equina, spinal cord, pons varolii, and lungs—were all of the same nature, viz. spindle-cell sarcomata. Further, they appeared to be pure spindle-cell growths, no other kind of cell entering into their composition. We have already remarked upon the fact, so unusual in intra-spinal tumours, that the growth in the dorsal part of the cord simply replaced the nervous elements of the cord, the shape of the cord being, nevertheless, still retained; so that no distinct outgrowth or tumour projection from the cord at the seat of the disease occurred, except the affection of the various nerves along which the new growth had extended. It may, in fact, be said that we had, for a distance of two and a half inches in the dorsal region, what was apparently at first sight a portion of the spinal cord, but in reality was that structure replaced

by sarcomatous elements. The growth extended along the cord, above and below, by growing along the outside of the blood-vessels, so that nearly every small arteriole at the periphery of the growth was surrounded by a mass of spindle cells, and its lumen was considerably narrowed from the pressure exerted by these from without. This mode of extension left traces of its previous existence in the older parts of the tumour, where the spindle cells were seen to be arranged in concentric groups, forming very small nests, the centre of which represented the narrowed, and in many parts completely obliterated, lumen of a blood-vessel.

At the extremities of the growth in the dorsal region, the nervous matter, which still existed between the masses of spindle cells arranged in groups round the blood-vessels, was very much degenerated, large accumulations of myelin being present at these parts; these myelin masses appeared to be gradually absorbed, and to be replaced by the sarcomatous elements. At some parts of this tumour in the dorsal region, and at the periphery of it, similar masses of myelin still existed to those which were found at the extremities of the growth, and it was the persistence of this myelin that gave the appearance described in the post-mortem report as resembling calcareous collections; there was, however, no calcareous matter.

The nerves issuing from the part of the cord which was replaced by the new growth, all suffered from an invasion into their substance of the sarcoma elements. The spindle cells had developed in the perineurium, surrounding and isolating the true nerve fibres, without apparently exerting very great pressure on those fibres, for even in the oldest parts of the growth the arrangement of the fasciculi of these nerve-roots was still distinct, but no axis cylinders could be recognised at any part of them; the absence of the latter, and the disappearance of the myelin, left a large number of small, more or less circular spaces, which had been previously occupied by the nerve fibres; the large number of these spaces in the sarcomatous infiltrated nerves, gave a hyaline appearance to the sections taken from these parts. The spindle cells of the tumour appeared to have simply replaced the perineurium, and

the true nervous elements, the axis cylinders and their myelin sheaths, had then become absorbed.

The appearance which these nerves presented on emerging from the affected portion of the spinal cord, was likewise again seen in the growth affecting the corda equina, only here it was seen on a larger scale, the growth having matted together the various nerves composing that structure. The spindle cells had here extended again between the separate nerves of the corda equina and between the various fasciculi of each nerve; but still the original arrangement of each nerve could be still recognised, for though the axis cylinders and their myelin sheaths had disappeared, the small spaces which they had occupied still persisted, surrounded on all sides by the spindle cells of the growth.

Those parts of the growth in the cord and in the corda equina which had developed most recently, were very rich in nuclei; but in the older parts these were far less numerous, and the growth had there taken on a uniform hyalin-fibroid appearance. With this exception, the tumours had not undergone any degeneration or further change with age.

There was nothing in the microscopic appearance of the growth in the spinal cord, or of that in the corda equina, to allow us to say positively which was the older of the two, and which consequently represented the primary one, but that in the pons was certainly of more recent development than either of the other two.

Although the pathological details collected after death do not allow us to solve this point, I think there can be very little doubt that the tumour in the corda equina was the primary growth, and the growth in the dorsal part of the cord was secondary to it; the pons tumour developing still later. It is highly probable that for the first eighteen months of his illness he was suffering from the growth in the corda equina alone, and it was only in the first month that he came under our observation that the tumour in the dorsal region began to develop, and that the complete paraplegia was brought about by the combined effect of the two tumours, and was not to be attributed to the tumour in the corda equina alone; that it was the development of this tumour in the dorsal region

which in great part accounted for the marked downward progress of the man when he was at the Cheadle Convalescent Hospital, and the rapid development of the complete paraplegia after his return to the Royal Infirmary. What speaks very strongly in favour of this view is, I think, the fact noted that soon after he became completely paraplegic, a patch of anæsthesia appears, not only over the left thigh, but also over the *costal margin* of the right side, a position which could not be accounted for by the tumour in the corda equina, but was probably due to the growth in the dorsal region, and possibly its extension along some of the sensory nerve roots from that portion of the spinal cord.

The spinal cord below the tumour in the dorsal region presented very well-marked and very advanced sclerosis of both lateral tracts, and also of Goll's and Burdach's column. The lateral sclerosis on either side was due to the interruption of the cord in the dorsal region by the tumour there; it was, in fact, a descending sclerosis.

The sclerosis of Goll's and Burdach's column on either side presented itself as a marked sclerosis, which extended on both sides from the posterior median fissure to the inner aspect of the posterior grey cornu, with no break of intervening healthy nerve tissue. This sclerosis we must regard as an ascending sclerosis from the effects of the growth in the corda equina. It is interesting as illustrating the fact that extensive lesions of the corda equina cause, not only a sclerosis of Goll's tract, but also of Burdach's as well. A considerable portion of Burdach's column, in the lower portions of the cord, being probably composed, either directly or indirectly, of fibres which proceed from the various nerves of the corda equina, and so consequently, when the latter is divided or interrupted by such a growth as we have in this case, sclerosis of that column results.

Above the tumour in the dorsal region there was well-marked evidence of ascending sclerosis, the column of Goll being very sclerotic in its whole vertical extent, and sclerosis could be traced as far as the posterior surface of the medulla. Immediately above the tumour, for a distance of about a quarter of an inch, there was not only a sclerosis of Goll's

tract, but also of Burdach's, exactly as we saw was the case below the tumour. We must consider this change in the posterior root zone as an ascending sclerosis; but it is not probable that it represents the effects of the growth in the corda equina before the tumour in the dorsal region began to develop, since probably the latter tumour grew, and practically divided the cord, before the one in the corda equina had completely cut off the nerves going to the lower extremities; at the same time we cannot forget that lesions of the great sciatic nerve alone have been followed by a sclerosis of Goll's column and of the posterior root zone. However, I should be much more inclined to attribute this sclerosis of the posterior root zone, which existed for a very short distance immediately above the tumour in the dorsal region, to the effects of that growth, which would cut off a large number of the fibres which would subsequently proceed upwards for a certain distance in Burdach's column: in support of this we may note the complete destruction of all nervous elements in both the spinal cord, and in the nerves which entered that structure opposite the seat of the new growth.

In the cervical region distinct sclerosis of the cerebellar tract also existed; this could be traced into the medulla, where it occupied a small tract in the restiform bodies. This is again to be regarded as an ascending degeneration.

From the position and extent of the cerebral tumour, we should have expected some evidence of descending change in the left lateral column; there was, however, no continuous tract of sclerosis, though some sections did show slight increase of the neuroglia in the lateral column of one side, and there was a small patch of sclerosis in the anterior pyramid of the medulla on one side.

The cavity existing in the cervical region of the cord, which in the post-mortem report is attributed to a dilatation of the central canal, was in reality to be so explained, as microscopic examination clearly proved. In the above region only one cavity existed, which occupied the median line, and was quite $\frac{3}{8}$th inch in transverse measurement; it was flattened from before backwards, and so measured much less in that direction. It diminished in size from above downwards, being

distinctly smaller in the lower cervical region (from which Fig. 2 was taken) than just below the medulla oblongata (Fig. 1). In the upper dorsal region two canals existed, both of much smaller dimensions than the single one above: one of these occupied a central position, and represented the dilated central canal; the other occupied the position of the posterior grey cornu of the right side, having quite replaced that portion of grey substance. These two canals extended downwards about one inch, and were succeeded by a single canal occupying a central position, and evidently the original central canal of the cord, but now dilated, for a distance of about another inch, below which the canal again became double for a distance of half an inch, the two canals being again here situated as the two were higher up. Then below these two canals we had only a single one, occupying a central position as far as the tumour in the dorsal region. Below that new growth the central canal presented but slight increase above the normal dimensions, and was a single channel occupying its usual central position.

Consequently we had in the greater part of the cord an apparently very much dilated central canal; but in the upper dorsal region at two places two canals existed, between which two points there was only a single centrally situated channel. The wall of the canal in the cervical region (Figs. 1 and 2) was much thinner than in the dorsal region, where two canals existed (Fig. 4), although in the former place it enclosed a much larger channel than in the latter, where the lumen of either was very small. Further, the wall of the canal in cervical region was composed of firm, fibrous tissue, evidently of much older date and more fully formed than in the dorsal part of the cord, where each canal was surrounded by a fibro-nuclear growth, which was really very rich in nuclei. At the same time it could not be considered to represent a tumour mass, a new growth such as a glioma, but undoubtedly was an inflammatory tissue, and was probably to be regarded as the result of pressure exerted from within the canal. The lining membrane of the canal or canals was always well-defined, and often had a corrugated sinuous outline, or, as it has been described by some authors, of a shirt-frill character,

and we could hardly conceive how such a canal, with so thick walls and such an internal limiting membrane, could have originated by any decay in a previously existing tissue; far more probable appeared to be the explanation, that the cavity first existed with a thin wall, and that the latter thickened and became stronger to support the increased pressure.

The canal in the cervical region was so large, that though occupying a central position, it involved to a large extent the grey cornua, especially the posterior, which had practically disappeared (Fig. 1); the anterior cornua, on the other hand, were comparatively little implicated; but in parts even they were much compressed and diminished in size. The wall of these canals was composed of a fibro-nuclear tissue: in the cervical region, where the canal was large and single, the fibrous element predominated, and the wall was not so thick as elsewhere; in the parts where there were two canals, the nuclear element was in excess, and the wall was much thicker (Fig. 4), while the lumen of the canals was less than in the case of the single cavity in the cervical region. Towards its inner aspect, the wall of the cavity often presented a corrugated, folded appearance, just like the elastic coat of an artery. No endothelial lining existed.

The pathogenesis of such cavities as these in the spinal cord is still very uncertain. Although a really large amount of attention has been given to the study of the subject, hardly any two authors will be found to agree as to the exact sequence of events which has led to their formation, and it is quite possible, in fact, from the recorded cases *probable, that the same explanation will not suffice for all instances of the morbid appearance.*

It is well that we should start with a clear idea of what we mean by the term syringomyelia, and what by hydromyelia. Simon, Leyden, and Ziegler separate congenital enlargements of the central canal under the term hydromyelia, from those cases where we get cavities formed from pathological causes subsequent to birth, and to which the term syringomyelia is applied. To the latter group would belong our present instance.

It is noteworthy that this affection is most commonly either

limited to the cervical or dorsal parts of the cord, or is most advanced there; also that the cavities which are formed are most frequently situated either in the posterior white columns, the posterior grey commissure, or in the posterior cornua, very rarely do they appear in the anterior portions of the cord, either in the grey or white substance.

Leyden[1] regards syringomyelia really as a consequence of congenital hydromyelia, attributing it to an interference with the closing the central canal, which is at first so large in the embryo. The position in which these cavities are found, chiefly in the posterior parts of the cord, would be held to support this view.

Westphal[2] and Simon[3] both attributed it to the development of a new growth, a tumour (and not inflammatory tissue) in the spinal cord with a subsequent degeneration of the centre of this, and the formation of a cavity or canal. According to their views, the cavity has nothing directly to do with the original central canal of the cord. Westphal, however, in a more recent paper,[4] shows that his former view must to a certain extent be given up, and that a certain relation of such cavities to the central canal must be acknowledged. And he says, the changes found in the cases most recently observed by him were doubtless congenital, and that we must assume that disturbances of development of the central canal ensued with cavity formation, accompanied by a tendency to the development of fibrous new growth. He adds, however, that "in other cases there seems to be a tendency to the development of another form of new growth (glioma, myxoma)."

Schultze[5] likewise holds the view, that these cavities owe their origin to a decay of proliferated neuroglia tissue, which may take its origin at various parts of the spinal cord, or in the medulla oblongata.

Hallopeau[6] regarded a central myelitis as the primary

[1] 'Klinik der Rückenmarkskrankheiten,' ii. p. 447; and Virch. 'Archiv,' vol. 68. Compare also Taylor, F., 'Trans. Path. Society,' 1884, vol. xxxv. p. 36.
[2] 'Archiv f. Psychiatrie,' vol. 1, 1874, pp. 30 and 109. [4] Ibid.
[4] 'Brain,' July 1883, pp. 149 and 165.
[5] Virch. 'Archiv,' vol. 87.
[6] "Contribution à l'étude de la sclérose diffuse périépendymaire," &c., 'Gaz. Méd.,' 1870, 30, 32, 34, 35.

lesion; a diffuse sclerosis ensuing round the central canal, and in consequence of degenerative changes which ensued in this inflammatory tissue, cavities were formed.

Langhans[1] attributes the cavities to a dilatation of the central canal of the cord, and the formation of diverticula from it, brought about by blood stasis, which stasis may be occasioned in various ways; in three of his four cases it was effected by tumours in the posterior fossa of the skull.

My own case I am inclined to attribute to dilatation of the central canal, and the formation of diverticula from that, but I am less inclined to explain that dilatation by a blood stasis occasioned by the tumour in the pons, than by an obstruction to the circulation of the cerebro-spinal fluid, and to a damming up of that fluid in the central canal of the cord, whereby great pressure was exerted, diverticula formed, and as a consequence of that pressure, a sclerosis ensued round the canals so arising, and that consequently the sclerosis was not a primary element in the case.

The most recent writer on the subject is Dr. Ed. Krauss,[2] who ably reports a very interesting case. He explains the syringomyelia which existed, as a consequence of proliferation of the neuroglia tissue, which first formed, as it were, a kind of glioma, and then the central part of this undergoing decay, cavitation resulted. After carefully considering the previous history, and the pathological appearances found after death in Dr. Krauss's case, I am much more inclined to think that the primary lesion there was a hæmorrhage into the upper part of the spinal cord, and possibly medulla, with a secondary descending sclerosis of the lateral column of the one side, together with the formation of a cavity, the greater part of which was due to a dilatation of the central canal of the cord, but that part of the cavity above might be due to changes ensuing in the hæmorrhagic focus itself.

Dr. Krauss believes that there had previously existed a syringomyelia which had caused no symptoms until those ensued quite suddenly (the patient waking up one morning

[1] "Ueber Höhlenbildung im Rückenmark als Folge von Blutstauung," Virch. 'Archiv,' vol. 85, p. 1.

[2] "Ueber einen Fall von Syringomyelia," Virch. 'Archiv,' August 1885, vol. 101, p. 304.

and finding the right leg and arm paralysed), and he is evidently of the opinion that the most probable explanation of the sudden onset of those symptoms was a hæmorrhage, which, as he remarks, is no seldom occurrence in the case of a glioma. For my own part, I can see no reason or necessity to assume, that before the hæmorrhage ensued such a proliferation of the neuroglia did exist, nor that there was before the onset of the symptoms, any syringomyelia. A far more rational view appears to me to be, that hæmorrhage into the spinal cord (rare as such lesion is) was the primary change.

As regards my own case, I think few could study the sections from which the figures were taken without coming to the conclusion, that most probably the sequence of events had been increased pressure within the central canal, dilatation of this (Figs. 1 and 2), formation of a diverticulum (Fig. 3) or outgrowth from the same, and eventually the establishment of two distinct channels (Fig. 4) with very thick walls.

From cases recorded, it is evident that syringomyelia may reach a very advanced stage without giving rise to any definite symptoms during life. In others it occasions marked atrophy of various groups of muscles, with or without sensory disturbances. It is doubtful whether, in our own case, any of the symptoms could be referred to the syringomyelia and to the sclerosis round the canals which existed. The grey matter was very extensively involved, as Figures 1 and 2 show; in some parts not only was the posterior (Figs. 1, 2 and 3), but also the anterior cornu of one or other side considerably affected (Fig. 3). It is hence possible that such a lesion might have been really the cause of the deficient chest movement, which was noted in the latter part of our patient's existence. It is certain that the position of the tumour in the dorsal region, and the involvement of the issuing spinal nerve-roots, would not account for this paralysis of the intercostals.

An interesting point also in our case was the existence in the anterior pyramids of the medulla, close to the anterior surface (Fig. 5 *a*), of a distinct and well-marked nucleus of grey matter, which was so large as to be quite easily visible to the naked eye. This nucleus commenced immediately above the

decussation of the pyramids, and ran upwards to close upon the lower border of the pons, a vertical distance of nearly ¾ inch. Quite at its upper part this nucleus divided into two, one occupying the position the whole did lower down, viz the anterior surface of the medulla, while the other and smaller half was situated at the side of the anterior median fissure, rather more posterior and internal than the above-mentioned half.

At its point of greatest size, which was at its lower part, this nucleus presented upwards of two hundred ganglia cells; these diminished in number towards the upper limit of the nucleus.

No nerves were seen coming off from the anterior surface of this nucleus, but from its deeper part, filaments of nerves ran into and were lost, in the deeper parts of the medulla. No connection could be traced between this nucleus and any of the other nuclei in the medulla (Fig. 5 *a*).

Another well-marked nucleus of grey matter existed at the posterior part of anterior pyramids immediately to the inner side of the hilum of the olivary body (Fig. 5 *b*). I specially mention the existence of these two nuclei, since the majority of our English anatomical text-books quite ignore their existence, and I possibly may hence be forgiven for having at first considered them to be but another item in making our patient a pathological curiosity.

Both of these nuclei are, however, recognised in the majority of the anatomical works of continental authors; they are, in fact, the nuclei arciformes of Henle,[1] or the anterior and posterior pyramidal nuclei of Kölliker, and the small pyramidal nuclei of Stilling.[2]

In our present case they were both exceedingly well developed.

The cerebral symptoms call for special mention in reference

[1] 'Handbuch der systematischen Anatomie des Menschen,' von Dr. J. Henle, 1868, Nervenlehre, p. 194.

[2] See also 'Allgemeine und microscropische Anatomie,' von W. Krause, III Auflage, 1876, I. Bd., page 413 and Fig. 242; also 'Zehn Vorlesungen über den Bau der Nervösen Centralorgane,' von Dr. Ludwig Edinger, 1885, page 121, Fig. 107. Also Ross 'On Diseases of the Nervous System,' 2nd edit., vol. i. p. 807: "The nucleus of the pyramid (internal parolivary body)."

to one point. The trophic lesions in the area of distribution of the right trigeminus were highly interesting, but what I would specially call attention to is, that though the post-mortem proved how complete had been the implication of the 5th nerve with the Gasserian ganglion of the right side, yet there had not been the slightest affection of taste on the corresponding side of the anterior two-thirds of the tongue. I repeatedly tested for this, since the case was under observation at the time when I became acquainted with Erb's views on the course, which the gustatory fibres supplying that part of the tongue took to the brain.[1]

Whatever may be the course of the gustatory fibres of the chorda tympani in the majority of cases, it is certain that in our case they did not pass with the 5th nerve on the right side. Although many cases are recorded of loss of taste over the anterior two-thirds of the tongue from disease of the 5th nerve, others have existed, and been put on record, where complete paralysis of that nerve left taste unaffected.

In conclusion, I would subjoin a few further references to the subject of syringomyelia:

NONAT: "Recherches sur le développement accidental d'un canal rempli de sérosité dans le centre de la moëlle épinière."—'Archives générales,' 1838, i.

JOLYET: "Sur un cas d'anomalie du canal central de la moëlle épinière."—'Gaz. Méd. de Paris,' 1867.

VIRCHOW: "Die Betheiligung des Rückenmarks an der Spina bifida und der Hydromyelia."—Virch. 'Archiv,' xxvii. 1863, p. 575.

LANCEREAUX: "Un cas d'hypertrophie de l'épendyme spinal avec oblitération du canal central de la moëlle."—'Mém. de la Soc. de Biol.' Paris, 1862.

SCHÜPPEL: "Ueber Hydromyelus."—'Archiv der Heilkunde,' vi. p. 289.

GRIMM: Virchow's 'Archiv,' 1869, vol. 48, p. 445.

NAUNYN and EICHHORST: "Ueber die Regeneration und Veränderungen im Rückenmark."—'Archiv f. exper. Pathol. und Pharmakol.,' ii. 1874, p. 225.

STRÜMPELL: 'Arch. f. Psychiat.,' x.

FRIEDREICH: Virchow's 'Archiv,' vols. 26 and 27.

[1] "Ueber den Weg der geschmackvermittelnden Chordafasern zum Gehirn," 'Neurogl. Centralb.,' vol. i., 1882.

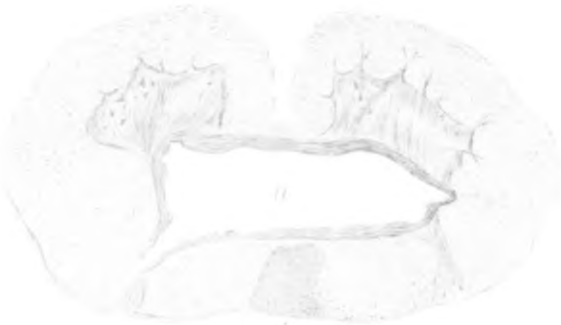


Fig. 1




Fig. 2

Fig. 3

Fig 3

T. Harris del. Entwistle & Co. lith.

PICK: 'Archiv f. Psychiatrie,' viii. p. 285
RIPPING: "Cystoide Degen. der Hirnrinde."—'Allg. Zeitschr. f. Psych.' vols. 30 and 32, 1874 and 1875.
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WHIPMAN, T.: 'Trans. Pathol. Societ.' vol. xxxii. 1881, p. 8.
KESTEVEN, W. B.: 'St. Barth. Hosp. Reports,' 1872, vol. viii. p. 15.
REMAK: 'Deutsche Med. Woch.' No. 47, 1884.
SCHULTZE: Virchow's 'Archiv,' vol. 102. 1885.

## EXPLANATION OF THE PLATES.

Fig. 1. From the extreme upper part of the spinal cord.
*a*. Extremely dilated central canal with comparatively thin fibrous wall.
*b*. Sclerosis of Goll's columns.

Fig. 2. From lower cervical region.
*a*. Dilated central canal, here smaller and with a thicker fibrous wall.
*b*. Sclerosis of Goll's tracts.

Fig. 3. Lower cervical region.
*a*. Formation of a diverticulum (*c*) from the dilated central canal.

Fig. 4. From dorsal region.
*a*. Two cavities (*a* and *c*) with thick fibro-nuclear walls.

Fig. 5. From the medulla.
*a*. Anterior pyramidal nucleus.
*b*. Posterior pyramidal nucleus.

# ON A CASE ILLUSTRATING THE CORTICAL NATURE OF EPILEPSY AND ITS RELATIONSHIP TO JACKSONIAN CONVULSIONS.

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SINCE Bastian first argued for the existence of cortical "perceptive centres," and since the researches of Ferrier have demonstrated that centres connected not only with the motor functions, but also with the perception of special sense-impressions occupy definite areas in the cortex cerebri, much has been done, both experimentally and clinically, to prove that all forms of epilepsy are cortical in their nature.

Among the many workers in this field of inquiry, Hughlings-Jackson stands out pre-eminent in having demonstrated by clinical and pathological research the connection of certain definite forms of epileptic attack, with coarse lesions of the motor areas of the cortex. But, though experiment renders it extremely probable that similar irritative lesions in the sensory areas of the cortex may also cause epileptic attacks, the clinical evidence on this point is still very deficient.

The following case is of considerable interest in this respect, since, in connection with a well-localised external injury in relationship to both motor and sensory cortical areas, two perfectly distinct classes of fits occurred—the one resembling in all essential features the motor seizures of Hughlings-Jackson, and the other corresponding to the ordinary epileptic attack.

For leave to publish this case I am indebted to Dr. Brakenridge, in whose ward it occurred.

William Heslop, aged 23 years, and unmarried, a brass-finisher by trade, but recently working as a labourer, was

admitted to Ward 32 of the Edinburgh Royal Infirmary on Oct. 11th, 1883, complaining of "fits," which had troubled him for four or five years.

### History.

1. *Family History.*—The patient is the eldest of a family of seven, four of whom are dead. All four died in infancy; in the case of one the cause was "water on the head," in the other cases the cause of death is unknown. The other surviving members of the family, a brother and sister, are strong and perfectly healthy. His father died in Morningside Asylum, suffering from general paralysis of the insane. He had been a very hard drinker. One of his father's brothers died of delirium tremens, but his father's other brothers and sisters are said to be free from disease. His cousins are all healthy. His mother is healthy, and there is no neurotic history in connection with her family.

2. *Personal History.*—The patient has always had a comfortable home and good food, and his work has never been excessive. He generally on Saturday night indulges freely in beer, but he is not a heavy drinker.

3. *Past illnesses and accidents.*—As a child he had measles and scarlet fever; but since then he never had any illness. There is no history of syphilis, or of convulsions during teething.

At seven years of age he fell over a stair, three stories high, and sustained a very severe fracture of the frontal bone, for which he was trephined by Dr. Gillespie. After the operation he made a rapid and complete recovery.

Five years ago, when working on board a steamer at Glasgow, an iron bucket, weighing about half a hundredweight, fell upon the right side of his head, inflicting a compound fracture of the parietal bone. He was stunned for a moment, but almost immediately regained consciousness. Before he could be got to the Infirmary he was again unconscious, and remained so for several days. He was under the care of Dr. McEwan, and I have been able to ascertain that the treatment was expectant, and that no head-symptoms developed. The patient himself states that a small piece of

bone was removed. He was not discharged from the Infirmary for eighteen months, during which time the wound on the side of the head remained unhealed. Three weeks after his dismissal his *present illness* commenced.

4. *Present illness.*—Walking along the street in Glasgow, he suddenly fell down in an unconscious condition; and upon recovering, he suffered for some time from severe headache. Since then he has been constantly liable to fits, which come on at irregular intervals, sometimes when he is awake, sometimes when asleep. If he is seized during sleep, the fit is generally much more severe.

Two kinds of attack are described:—

In the first, consciousness is almost at once and completely lost; while in the second it remains undisturbed, either throughout the whole fit, or for a considerable time after its commencement.

In connection with seizures of the first type, all the patient can tell is that they are usually preceded by a very distinct and somewhat complex aura. He first experiences a sensation of heat in the left side of his face, and almost simultaneously he sees a ball of fire before the left eye, while at the same time he hears a singing sound, but whether in one or both ears he is unable to say. His mother describes these fits as very violent, and from her description they appear to resemble very closely in all particulars ordinary epileptic seizures.

Of the second type of convulsion the patient gives a very clear account. The fit commences with a twitching of the whole upper lip, or of one side of it, sometimes the right, sometimes the left. Ultimately, however, the left side is most markedly affected, and the mouth and cheek are drawn over to the left side and twitched violently. The muscles concerned in flexion and extension of the left elbow-joint are next involved, first becoming rigid, with the arm fixed in a position of semi-flexion, and then passing into a state of clonic spasm. The next step is the flexion of the hand on the forearm, and then the shoulder-muscles are affected, first with a tonic and subsequently with a clonic spasm, while the head is twisted over to the left side, and the whole body is bent to the left. Consciousness is generally lost at this stage of the

attack, but the motor spasm may first extend over to affect the right arm. As soon as the spasm reaches the right side, consciousness is invariably lost, and it is impossible to trace the further progress of the fit. He states that upon recovering consciousness, he finds the left arm still jerking. After the fit he suffers from severe frontal headache, equally intense upon both sides and throbbing in character.

The convulsion does not always go so far as above described; frequently it never passes beyond the arm, and consciousness is then retained throughout.

### State on Admission.

Weight, 8st. 9lb.; Height, 5ft. 5in. He is a well-developed, fairly strongly built young man, of fair complexion, and with a large brachycephalic head. The eyes are somewhat deeply set, the pupils large and equal, the iris is of a lightish-grey colour. The conjunctivæ are clean. The features are small and well-formed, and the expression is intelligent.

The left side of the forehead is more prominent than the right, but the supra-orbital ridge is, at its outer extremity, obliterated. At a point 2½ inches from the middle line, and 1¼ inches above the eyebrow, there is a well-marked rounded protuberance; and running upwards and inwards from this is a ridge 2 inches long. Under this ridge is a depression 1¾ inches long by 1 inch broad at its widest part. It is ovoid in form—the large end of the ovoid being directed downwards and outwards. Two inches above the root of the nose is a crescentic scar, with its concavity directed upwards, and this terminates at its left extremity in a triangular somewhat depressed cicatrix. Over the whole of the ovoid depression very marked pulsation may be observed, and when the patient stoops, not only is this pulsation increased, but a well-marked bulging becomes visible. A bridge of bone passes horizontally across the middle of the ovoid, but above and below this a depression, which is soft and yielding to the touch, may be felt. The median triangular patch also pulsates and bulges when the patient stoops. The crescentic cicatrix covers a depression, having its deepest part at its right extremity. Uniting the right angle of the triangle with the

upper and inner end of the ovoid, a narrow linear depression may be felt.

On the right side of the head, 4½ inches above the tip of the mastoid process, is a cicatrix, uncovered by hair and partly adherent to the subjacent parietal bone. It covers a linear depression about ¼ inch broad, which extends, from a point ½ inch behind a perpendicular drawn through the mastoid process, upwards and forwards for 2 inches to a point 4 inches above the tragus of the external ear. The depression is deepest and broadest where a vertical line from the external auditory meatus intersects the scar.

My friend Mr. Hare kindly examined the position of this scar for me, and from a long series of observations—the results of which he has since published,[1]—he believes that this depression must be entirely behind the fissure of Rolando, and that it must pass over the lower part of the ascending parietal convolution, and run downwards and backwards over the posterior part of the fissure of Sylvius.

### Nervous System.

1. *Sensations.*—He suffers from headache and dimness of vision for some time after his fits.

2. *Sensibility.*—1. Common sensibility, thermal sensibility, &c., are unaltered. 2. *Special Senses.*—Pupils are equal, widely dilated, and respond only slightly, but equally to light and to accommodation. The field of vision and visual acuteness are unaltered.

*Ophthalmoscopic examination.*—Both eyes are slightly myopic—the media are clear; in both there is a well-marked deep cupping of the disc. The laminæ cribrosæ are well marked. In the left eye are marked signs of old neuritis, probably connected with injury to the eye from his first accident.

Taste, hearing, and smell are normal. The muscular sense is also normal.

3. *Motor Functions.*—The organic reflexes are normal. During the fit the patient does not lose control over the bladder and rectum. The tendon-reflexes are normal, and the cutaneous reflexes are all fairly marked. Voluntary movements are

[1] 'Journal of Anatomy and Physiology,' vol. xviii. p. 174.

in all respects normal. Co-ordination is perfect; and the electrical irritability is unaffected.

4. *Cerebral and Mental Functions.*—The patient sleeps well. His mental faculties are well developed; altogether he is a most intelligent, clear-headed fellow, fond of reading and of various games.

All the other systems are normal.

## Diagnosis.

The seizures first described correspond in all essential particulars to Hughlings-Jackson's classical description of that form of epilepsy named after him; and that they are really of this nature is rendered certain by their occurrence in connection with a well-defined traumatic lesion in the region of the cortical centres related to the muscles first implicated in the attacks.

Unfortunately, after his admission to the Infirmary, the patient remained free from these more purely motor seizures, so no opportunity was afforded of studying their onset, and of thus definitely localising their starting-point.

The patient's own statement, confirmed by that of his mother clearly shows the lips were first implicated. But this is not sufficiently definite. For it must be remembered that the labial centres occupy no small part of the motor area of the cortex.

Mr. Hare's careful measurements, however, which were found post-mortem to have yielded extremely accurate results, indicated that the injury did not extend in front of the fissure of Rolando; that it implicated the lower part of the ascending parietal convolution, part of the angular gyrus and part of the superior temporal convolution; that, in fact, the only motor area involved was No. 11, that centre connected with retraction of the angles of the mouth.

In all probability the clinical study of this point would not have yielded very accurate results, since the close physiological relationship of all the labial centres, and the consequent almost simultaneous implication of these, would have rendered it impossible to have detected in which the nerve explosion really commenced.

The characters of such Jacksonian seizures are now so generally known to the profession that it is needless here to enter more fully into the various phases of the attacks.

It is of some interest, however, to compare the march of spasm on the body with its course over the cortex cerebri. Centre 11 being first affected; centres 7, 8, 9, and 10, are next implicated; while 6, the centre for the bicipital movements, and *a*, *b*, and *c*, for the movements of the hand and wrist, are subsequently involved. The more remote centres, 12, 5, 4, 3, 2 and 1, are last implicated, just before or just after the spasm passes to the other side of the body.

But besides these motor attacks, our case presents for our consideration seizures having different and graver manifestations; and the question which we must now consider is whether these are due to an idiopathic epilepsy; or whether, on the other hand, they owe their origin to the same injury which we have been led to consider the cause of the minor seizures.

The evidence in favour of true epilepsy seems at first sight very strong. We have the patient's family history. His father died insane, and an uncle suffered from delirium tremens.

But that it is not a true idiopathic epilepsy—that it is not what would be called by Nothnagel a primary epilepsy—that is, an epilepsy due purely to inherent and inherited causes, is I think manifest from the very definite connection of these fits with the head-injury; though of course it is extremely probable that our patient inherited from his progenitors brain-cells so unstable as to render him peculiarly susceptible to any exciting cause of epilepsy.

But granting that this is a secondary epilepsy dependent upon the head-injury, the question now arises—do these more typical fits owe their origin to the same physical condition with which we have connected the Jacksonian seizures; or must their cause be sought elsewhere?

This blow on the head might have induced such seizures in different ways.

Firstly, we might have here a clinical example of one cause of epilepsy which Westphal has demonstrated in guinea-pigs.

[1] 'Berliner Klinische Wochenschrift,' 1871, No. 31.

He found that if the head of the animal be struck lightly, a convulsion may be induced. This rapidly passes off, and leaves the animal in an apparently perfectly normal state of health for a period of some weeks, when an epileptic condition appears, fits occurring either spontaneously, or in connection with an epileptogenous zone. Post-mortem examination of these guinea-pigs reveals numerous small hæmorrhages among the fibres of the white substance of the spinal cord and upper part of the medulla.

The bearing of these experiments upon the case before us is so evident as to need no further remark from me. Unfortunately, the clinical evidence of the occurrence of such fits is small, unless in connection with embolism.

Secondly, Brown-Séquard has now most fully and satisfactorily demonstrated the possibility of inducing in guinea-pigs epileptic attacks, and an epileptic state lasting for a considerable length of time, by section or injury of peripheral nerves, either sensory or mixed. He found that, soon after section of the nerve—the sciatic was generally used—over the same side of the head and face an area of increased superficial reflexes appeared. This he called an epileptogenous zone. These reflexes gradually increased till a complete local spasm might be induced, and after some few days the spasm affected the whole of one side of the body. In the course of a month or six weeks, instead of unilateral spasm a well-marked epileptic fit resulted from irritation of this area.

Cases in man of this form of secondary epilepsy have occurred, though, as a rule, without the formation of epileptogenous zones, and it is a matter of no little interest to note that such cases are usually connected with an injury either to the sciatic or to the fifth cranial nerve. To quote Nothnagel's words:—"The lesion generally consists in external traumatic agencies, acting upon the trunk, branches, or cutaneous distribution of these nerves, less often on pressure due to tumours." In our case the area of distribution of the fifth cranial nerve is thus injured.

Against the supposition that our case is of this nature must be set the fact that the aura, when it occurs, is of the nature of some peculiar sensation in the area of the affected nerve. Here, however, we have a distinct aura in quite another region.

I believe that in our case we have to deal, not with an idiopathic epilepsy, but with an unusual form of secondary epilepsy, having its starting-point in the special sense-areas of the cortex.

I have been led to this conclusion firstly, by the localisation of the external wound in relation to the cerebral convolutions; and secondly, by a study of the aura epileptica.

I have already stated that Mr. Hare, from these measurements, is of the opinion, that the fracture must have passed over the lower part of the ascending parietal lobe, and extended across the posterior part of the Sylvian fissure—that it passes over area 11, over part of 13, and part of 14. Now, as is well known, area 13 has been proved by Ferrier to be the visual centre, while 14 has been similarly demonstrated to be the auditory centre; so that, in case of any meningeal thickening or other lesion in relation to the inner surface of the fracture, we must conclude that centres 13 and 14 are affected in the same manner as 11—the motor centre which we have seen participating in the commencement of the Jacksonian attack.

Secondly, the auræ, as described by the patient, are—first, a feeling of warmth over the left cheek; secondly, a ball of fire before the left eye; and thirdly, a singing in one or both ears.

Now these special sense-auræ are just what, from Ferrier's researches, we should expect in the case of an explosion of nerve energy in the ganglion cells of the visual and auditory centres, and that these cells are in all probability in an unstable condition we have already shown. The meaning of the thermal sensation is more difficult to explain, from the fact that centres connected with such sensation have not yet been localised.

I think then that we are justified in concluding that these, as well as the more purely motor fits, owe their origin to some gross lesion in connection with the fracture.

It is not difficult to understand how one exciting cause may act upon the cells of the sensory area; while another, probably starting from a different source, will more distinctly affect the motor areas.

That a sudden discharge of nerve energy, occurring in con-

nection with the sensory areas of the brain, should cause a more generalised convulsion than a similar discharge in a group of motor cells, is obvious; firstly, from the very intimate connection of each sensory area with many different motor centres, as is well shown by the very complex series of movements which supervene upon the irritation of such a sensory area; and, secondly, from the fact that nervous discharges, starting from the sensory cells, pass in a great measure towards the motor cells; that, in short, while the natural course of a discharge from a motor cell is outwards to the periphery, the course of a sensory discharge is towards motor cells, upon the action of which the natural manifestation of the sensory impression depends. Thus, once given the initial sensory disturbance, it is pretty obvious that the fit must become general, and cannot remain localised, as was the case in many of the motor attacks.

This diagnosis, arrived at during the life of the patient, was confirmed post-mortem.

Illness prevented my studying the progress of the case; but I have ascertained that after his first month in hospital he remained entirely free from fits; that he grew stupid, lethargic and heavy; that his sight became impaired, and that his general health steadily deteroriated. He went home for a short time in January 1844, but was readmitted almost immediately, and died on January 24th.

### Autopsy.

On January 25th, Dr. Byrom Bramwell made a post-mortem examination, the report of which I append.

*External Appearances.*—Length 55″, circumference at shoulders 37″; emaciated, pale; pupils equal and contracted; rigidity moderate; lividity slight; putrefaction commencing. A cicatrix, ½″ long, above left eyebrow (its middle); another in the middle line, 1½″ above the root of the nose. A well-marked depression at the seat of the first-mentioned cicatrix. Another depression, 3″ above and 2″ behind the right ear (external auditory meatus).

*Brain.*—Fracture of the frontal bone extended from the depression above the eyebrow up to middle of forehead; bone

absent at seat of depression, a hole the size of a sixpence bridged over by membrane. The dura mater and arachnoid are firmly adherent to the perforation in the frontal bone. At the seat of the depression behind the right ear was an old fracture. A needle could be passed through the narrow fissure at the bottom of the fracture, which was bridged over by membrane. The dura mater is adherent to the inner surface of the bone at the point of the external fracture; but no lesions of the arachnoid or convolutions here.

The tip of the left frontal lobe was destroyed by a cyst, and was fixed to the inner table of the skull at the seat of fracture in the frontal bone by the adhesion mentioned above. The cyst was about the size of a walnut. Surface of the brain otherwise quite normal. Weight 3 lbs.

Right optic disc and retina normal.

The pericardial sac contained 1 oz. of blood-stained serum; one or two punctiform hæmorrhages in posterior surface of the pericardium. The heart weighed 7½ oz., and was healthy. Both pleural sacs dry. Right lung weighed 1 lb. 7½ oz., very much congested, especially at lower lobe. Some œdema of upper lobe. Left lung weighed 14½ oz., much congested. Spleen weighed 4 oz., congested. Liver weighed 2 lbs. 3½ oz., congested, fatty and decomposing. Kidneys; right weighed 5 oz., congested; left, 3½ oz., in similar condition. Stomach, patches of congestion. Intestine normal.

Dr. Clemon, Dr. Brackenridge's then resident physician, was able, post mortem, to confirm Dr. Hare's measurements in regard to the locality of the fracture in relationship to the Sylvian fissure and the neighbouring convolutions.

I must add to this report the fact, that at the seat of the fracture of the right parietal, the inner table of the skull showed marked signs of inflammatory action, while the adherent dura mater was considerably thickened.

### Commentary.

Satisfactory as is the post-mortem in regard to the localisation of the lesion, it demonstrates the existence of a disturbing element unrecognised during life. The presence of a large cyst in the frontal lobe of the left side, connected with the

fracture sustained during early childhood, at once introduces the question of how far this was connected with the development of the epileptic seizures. I believe we may safely exclude this possible factor. For a careful analysis of the numerous recorded cases of lesions of the frontal lobes[1] shows that their connection with epileptic attacks of such a very distinct character is unknown, though undoubtedly epileptic fits may be caused by such lesions, just as they may be caused by lesions in any part of the nervous system.

Again, the development of these fits in such direct relationship to the later injury points very strongly to its causal connection with them, especially if we take into consideration the nature of the aura.

Perhaps the most interesting point demonstrated by the post-mortem is the absence of any marked change in the cortex cerebri at the seat of the fracture. It is greatly to be regretted that the preliminary treatment of the brain with too strong alcohol rendered it friable, and unsuited for microscopic examination. The inner surface of the parietal bone showed well-marked signs of inflammatory action, while the dura mater was distinctly thickened.

Other cases of epileptic attacks connected, as demonstrated during life by the aura, and post-mortem by the position of the lesion, with irritation of the special sense areas are on record;[2] but I have failed to find any case in which such a lesion produced two such totally different forms of seizure.

Such a case, connecting as it does the simple Jacksonian attacks with the more complicated manifestations of true epilepsy, is of very great interest and importance.

All evidence, clinical and experimental, points to the following conclusions:—1st, that the starting-point of all epilepsies is in the cortex; 2nd, that pure nervous explosion, without the intervention of vaso-motor changes, is sufficient to account for all the phenomena of the attack.

The second point has been fully treated by Gowers in his work on Epilepsy.

It is in connection with the former point that such cases as

[1] M. Allen Starr, M.A., M.B., 'Journal of Medical Science,' April, 1884.

[2] Starr, *loc. cit.*

ours are of special interest, affording, as they do, strong evidence in opposition to the medullary theory, still so ably supported by Nothnagel and others.

At first sight the experimental evidence upon which this theory is built appears extremely strong; but when viewed in the light of more recent research, it loses much of this apparent strength.

Undoubtedly the researches of Nothnagel have demonstrated beyond dispute the existence *in the rabbit* of an area in the pons Varolii and medulla oblongata, irritation of which causes general convulsions[1]; and at the same time have shown that the action of this centre is independent of the higher part of the brain—that convulsions do occur when the brain above has been removed. But Rosenbach, who has recently studied this subject,[2] contends that in dogs such fits do not manifest the characteristics of the true epileptic attack. And it must also be remembered that from experiments on the very undifferentiated brain of the rabbit it is hardly justifiable to draw conclusions in regard to the vastly more complicated and differentiated human brain.

Again, Nothnagel has not availed himself of the most valuable clinical method of research which we owe to Hughlings-Jackson—the careful study of the aura epileptica, the earliest symptom of the attack.

All evidence goes to prove that the perceptive centres are situated high up in the brain. Now, the aura epileptica is in the vast majority of cases of the nature of a perception of a sensation, and is not merely a reflex expression of such a sensation—such as would be produced by a nervous discharge commencing in the lower centres. Further, according to Gowers, in one-fifth of all cases in which an aura occurred this was of the nature of a special sense-warning, and the researches of Ferrier and all pathological evidence go to prove definitely that the centres connected with such perceptions are situated in a specific area of the cortex. When, on the other hand, the aura is of the nature of some localised muscular twitching or contraction, no doubt in regard to the cortical position of its starting-point can be entertained.

[1] Virchow's 'Arch.,' vol. xliv. p. 1. [2] Ibid.

Those cases in which no aura occurs cannot be cited as supporting the medullary theory, for we must remember that the absence of this warning is in all probability due to an inherent general weakness of the nerve-cells, allowing the nerve explosion to pass so rapidly from one centre to another that consciousness is lost before time is allowed for cognition of the initial perception.

Again, each attack deteriorates not only the group of cells first implicated, but also all secondarily involved, so that in time these will offer so little resistance to the spread of the attack, that the fit will very rapidly become sufficiently general to abolish consciousness.

By some the so common form of aura apparently connected with the pneumogastric nerve is considered as indicative of the primary affection of the medulla; but, as we have already pointed out, even these so-called organic auræ are of the nature of true perceptions, and therefore indicate the implication of the higher parts of the brain.

So much for the clinical evidence in regard to the nature of the epileptic attack, which certainly seems to render it highly probable that the starting-point of the fit is in the cortex cerebri. And this evidence is fully confirmed by the recent experimental research of Rosenbach.[1]

This research is of such importance, and appears to have received so little attention in this country, that I do not hesitate to give a brief *résumé* of the main results obtained by this observer. His experiments were conducted on dogs, and yielded the following result—

### A. *Motor Areas.*

1. Electrical irritation of a cortical motor area causes a well-marked epileptiform convulsion in the muscles corresponding to this area; and this convulsion passes by a definite route to other groups of muscles—the route corresponding to the proximity of the various motor areas of the brain.

2. On removing the electrodes, the attack passes off in the usual manner of epileptic attacks, the tonic spasm becoming clonic, and ending in paralysis of the muscles affected.

[1] Virchow's 'Archiv,' Bd. 97, S. 369.

3. It is difficult to regulate the current so as to confine the attack to one side of the body. When the median line is passed, the hind limb is the part first affected. When the centre for the hind limb is irritated, the attack frequently at once passes over to the opposite side.

4. At the height of the attack consciousness is lost.

5. After the fit one of two conditions may remain—

*a*. Post-epileptic paralysis.

*β*. Post-epileptic psychical irritability.

6. For a few minutes after the cessation of the attack, the centres respond extremely readily to irritation—a second attack being readily induced. But after from ten to fifteen minutes an increased resistance in the cells is to be observed.

B. *Non-Motor Cortical Areas.*

7. Irritation of non-motor parts of the cortex—posterior part—causes at first no convulsions, but after a latent period a general convulsion ensues. I need not point out how directly this bears upon the case we have had under consideration.

C. *Influence of Destruction of Cortex.*

8. Destruction of the motor areas on one side prevents irritation of the non-motor parts from inducing an attack. And irritation of the non-motor areas of the opposite side causes a general convulsion, confined to the side opposite the irritated area.

9. Any great destruction of the motor areas during an attack stops the convulsions in the muscles supplied.

10. A convulsion may be caused by irritation of the white substance from which the grey matter has been removed, but the convulsions stop without any clonic stage whenever the electrodes are removed. The convulsions induced do not tend to become general, and cannot be caused unless the current is stronger and only a small portion of the cortex has been removed. Irritation of the white substance lying below those parts of the cortex which are not motor causes no attack.

D. *Irritation of Medulla Oblongata.*

11. Irritation of the medulla oblongata causes a well-marked general muscular spasm, but no true epileptic attack.

E. *Influence of Bromide of Potassium.*[1]

12. On administration of bromide of potassium in large doses, it is almost impossible to induce an attack by irritation of the cortex, while irritation of the subjacent white substance causes spasms as in the unpoisoned dog.

He concludes—

1st. The convulsive attacks which are induced in dogs by electrical irritation of the brain are the result of stimulation of the cortical centres, and present, according to the source of irritation, the greatest similarity to the so-called cortical, or to the idiopathic epilepsy of man.

2nd. That between the so-called cortical and idiopathic epilepsy there exists—in their pathogenetic aspects—no real difference; but from the facts that the first is really a symptom, the result of organic brain affection, and in its clinical course is not identical with the latter, it must be kept distinct from the latter.

3rd. The convulsive attacks of idiopathic epilepsy, as well as the attacks of petit mal, are the effects of a primary diseased condition of the cortex cerebri.

4th. The multiple character of the picture of the epileptic attack depends upon the different manner and degree in which the pathological cortical irritation, which is the basis of the epileptic attack, spreads.

5th. The theory which places the starting-point of the epileptic attack in the centres of the medulla oblongata and Pons Varolii—is in opposition to the clinical features of the attack, and is not based on sufficiently sure facts even in its relation to the elucidation of the epileptic attack.

Such experimental evidence, especially when supported by the older observations of Luciani[2] and of Bubnoff and Heiden-

[1] 'Neurol. Centralbl.,' 1884, No. 2.

[2] 'Sulla patogenesi dell' epilessia; Rivista sperimentale di Freniatria,' &c. 1878, iv.

hain,[1] is of the greatest possible value, and may be said to demonstrate beyond dispute the cortical nature of all epilepsies.

The many transitional forms of epilepsy uniting Jacksonian convulsions with the idiopathic disease are of importance, as still further bearing out the experimental and clinical evidence of the cortical nature of all forms of the malady.

On the one side we have cases such as Heslop's, where the disease is apparently due to direct pressure on, or at least to direct irritation of the cells themselves. In this class we must also place many cases of epilepsy from syphilitic tumours, and perhaps a few post-hemiplegic cases of epilepsy.

Very closely connected with this class we have another well-marked group of cases connected with tumours in the *white* substance of the hemispheres, but not directly involving the ganglionic parts of the brain. These tumours appear to produce changes in the ganglion cells indirectly through nerve fibres upon which they press. I need not here adduce evidence—now so well known to every one—that such changes are continually brought about in cells through the action of nerve fibres, which are, after all, but continuations of the nerve cells themselves.

Not far removed from such cases are the experimentally induced epilepsies caused by striking the heads of guinea-pigs in which small hæmorrhages among the nerve fibres appear to set up the epileptic state; and in close clinical relation to these experimentally induced fits are most of the cases of post-hemiplegic epilepsy, which apparently owes its origin, in the vast majority of cases, to the presence of small extravasations in the neighbourhood of areas which have had their blood-supply interfered with by embolism.

Passing now outside the encephalon, we have Brown-Séquard's epilepsy from section of the spinal cord and of nerves, irritative changes apparently spreading upwards along the nerve trunks to affect ganglion cells connected with them; and, closely corresponding to these again, we have the so common "fits" of childhood, which, though at first connected with difficult teething or obstinate enteritis, pass sometimes into true epilepsy.

[1] Pflüger's 'Arch.,' Bd. xxvi.

Most closely connected with true idiopathic epilepsy are those cases of patients who, having inherited a weak and mobile nervous system from their parents, yet remain healthy till some exciting cause sets up an epileptic condition. And it is interesting to observe that by far the most common cause in these cases is a sudden fright. The very close relationship of fear to certain muscular movements—the outward manifestations of fear—is well known, and renders it readily intelligible that a fright might so act on a weak set of ganglion cells as to establish in them an epileptic habit.

Lastly, we have true idiopathic epilepsy occurring in the children of epileptics—a set of cases exactly corresponding to the epileptic young begotten by Brown-Séquard's epileptic guinea-pigs.

# ON A CASE OF AMNESIA.

BY E. A. DINGLEY, M.D. (LOND.).

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I HAVE submitted the notes of this case of Amnesia to my friend and former teacher, Dr. Charlton Bastian. He agrees with me in regarding it as one of great interest, and as belonging to that category which has been described by him as due to "damage to commissures between the auditory and the visual word-centres." In his work on "The Brain as an Organ of Mind" (3rd ed., p. 642) he has recorded a case of amnesia, in which some of the principal defects seemed to be due to a lesion producing functional inactivity of the two sets of commissural fibres naturally existing between the auditory and the visual centres. Two or three pages further on he refers to an important case published by Dr. Broadbent,[1] in which there appeared to be damage to one of these sets of commissural fibres only (viz. the visuo-auditory). Many of the peculiarities of the present case are to be accounted for on the supposition that here, amongst other things, we have to do with some damage to the other set of commissural fibres between these centres (viz. the audito-visual). My case is therefore the complement of that published by Dr. Broadbent.

Thus, in Dr. Bastian's cases the patients could neither read aloud, nor write from dictation; in Dr. Broadbent's case the man wrote well from dictation, but could not read aloud; in the present case, the patient could read aloud fluently, though he could not write from dictation.

In regard to the fluency with which my patient read aloud, it will be well to remember also a remarkable case recorded by Dr. Hertz.[2] "This patient," says Dr. Hertz, "was able to articulate distinctly any words which either occurred to him

[1] 'Med. Chirurg. Trans.,' 1872 (Case viii.)

[2] 'Psycholog. Mag.,' vol. viii.

spontaneously, or when they were slowly and loudly repeated to him. He strenuously exerted himself to speak, but an unintelligible kind of murmur was all that could be heard. The effort he made was violent, and terminated in a deep sigh. On the other hand, *he could read aloud with facility.*"

The patient whose case is here recorded was admitted into the Wolverhampton and Staffordshire Hospital, under the care of Dr. Millington, and I have to thank him for allowing me to publish the following notes.

John C., æt. 56, a brewer's drayman, was admitted on March 27th, 1884, complaining of having had a "stroke." He was rather more intelligent than men in his position usually are, and could read and write well, and keep simple accounts. He had suffered from attacks of gout for some time, but had never had any other illness. On March 25th he complained of headache, but went to work, and seemed in fairly good health. On March 27th he went out to work as usual, but came home at 8 A.M. "looking strange," and got into bed with his clothes and hat on. After a time he got up, and walked about the house in a "delirious" condition. He could speak plainly, but his right arm was weak, and he dragged his right leg slightly.

*On admission.*—He was found to be a strong, healthy-looking man; his temperature was 99°, and his face flushed. He complained of frontal headache, and of a "giddy feeling." He was conscious and rational, but his mind seemed somewhat confused and dull; the functions of the cranial nerves were carefully tested, and were all found to be normal except the facial, which was markedly paralysed on the right side. He protruded his tongue without any deviation. His speech was thick and slow; he only answered questions, and did not volunteer any statements.

Deglutition was normal.

Both arms could be moved, but there was marked weakness on the right side.

He could walk, but the right foot was slightly dragged. Sensation was not affected.

As regards reflexes, his pupils reacted well to light, and also

to accommodation, and were equal; the epigastric and plantar were well marked, but the cremasteric was absent on both sides. Knee-jerk could easily be obtained in both legs, but not ankle-clonus. There was entire control over the sphincters. His arteries were rigid and tortuous, and his pulse had rather a high tension. The heart was slightly hypertrophied, but no murmurs could be heard. The arcus senilis was well marked. The digestive and respiratory systems were quite healthy. Urine, specific gravity 1012; no albumen.

*March 28th.*—Patient is less intelligent than he was yesterday, and even partly unconscious; he recognised his master, but said nothing to him, and had a very dazed appearance.

*March 29th.*—Patient is semi-conscious: he says "Thank you" to every question. He does what he is told, but seems very confused and dull. He talks incoherently in his sleep. His right arm is now as strong as the left, but the right facial paralysis remains well marked.

*April 3rd.*—During the last few days patient's intellect has cleared up considerably; he says a few simple words, and appears to comprehend pictures. He sometimes complains of headache, localised about the left temple and left side of the forehead.

*April 5th.*—Patient can recognise pictures, but he cannot name the objects represented; he uses circumlocutory phrases; as, when shown a picture of a camel, he said, "Egypt long way." *He can copy writing, and can write from dictation his own name and such words as "cat" and "dog," but not more difficult ones.* He attempts them, but only makes a scrawl, though he occasionally gets the first letter right.

*April 10th.*—Patient is still improving; he can name a few simple objects. His grip, as tested with a dynamometer, is 50 on the right side, and 45 on the left.

*April 15th.*—Patient is still making progress—he can now read aloud easy words, but stumbles over the longer ones.

*April 26th.*—Patient *can now read aloud very well;* he can copy writing and easy drawings, *but cannot write from dictation any better than on the 5th inst.* He is still unable to name the most common objects.

*May 3rd.*—Patient can now write a very fair letter asking

leave to walk in the garden, but cannot write the same words from dictation.

*Remarks, with further details concerning Patient's condition two months after the attack.*

This case is interesting, as being one of those which can be easily explained by Dr. Bastian's theory of Amnesia as set forth in his work on 'The Brain as an Organ of Mind' (3rd ed. pp. 600–690), to which and to his clinical lectures I am indebted for my information on this subject. Before considering the morbid condition present in this case, it will be advisable to describe the (theoretical) normal condition.

In our brains we may be legitimately supposed to have two centres, or cell and fibre mechanisms, which are specially concerned with the expression of our thoughts by speech and writing. The more important of these is called the "auditory word-centre," and in it are stored up, if we may so speak, the remembered sounds of words. So that when we wish to express our thoughts, the necessary words are revived in the auditory word-centre and are then uttered. That this is so, is evident from a study of the process of learning to speak; for during this period the child learns to associate, and does after many efforts associate, certain sounds which represent the name of an object with the object itself, and, later on, with the idea or remembered impression of that object. This theory is also endorsed by the fact that deaf children, who never hear word sounds, never reproduce them in speech.

The visual word-centre, in like manner, is concerned with the form or shape of the words and with their reproduction in writing. This I believe to be part of a larger "form" centre, which comprises and reproduces all visual impressions of form, size, colour, &c. That there is such a centre is, I think, indicated by the fact, that when we are trying to reproduce a sketch, we call up in "our mind's eye," as the saying is, a picture or remembered impression of the object, and copy this as we should copy the actual thing if it were present. Again, when we are in doubt about the spelling of a word, we often write down one or two variations and see which harmonises

most with our previously received impressions stored up in the visual word-centre.

Each of these word-centres may be stimulated to action in three different modes, viz. (1) by volitional impulses, as in spontaneous speech; (2) by associational impulses coming from the other centre, as in reading aloud and writing from dictation; and (3) by sensory impulses coming from the ear or eye, as the case may be, when repeating words or when copying them from a book. Each centre must, therefore, be supposed to be in relation with three ingoing sets of fibres.

In addition, each centre is furnished with outgoing fibres to the other centre; and with others going, in the case of the auditory word-centre, to the nerves supplying the speech muscles; and in the case of the visual word-centre, to those supplying the muscles used in writing: but before reaching these outgoing nerve fibres, the impulses are supposed to pass through a centre called the "kinæsthetic" centre, in which the sensations associated with different movements are received, and whose activity is supposed to help to regulate the execution of future movements of all kinds, including, of course, those concerned with speaking or writing.

Thus the auditory word-centre furnishes us with the words which we require in order that we may express our thoughts; and this, together with the kinæsthetic centre, furnishes, and in part regulates, the impulses to the various muscles concerned in speaking the words. In a corresponding manner, the visual word-centre would co-operate with its related part of the kinæsthetic centre, for the execution of the movements concerned in the writing of words.

As regards the relative importance of the auditory and the visual word-centres, the auditory evidently comes first, not only because speech is far more common than writing, but also because it seems likely that in health words are recalled in the visual word-centre only through the auditory word-centre; or if not entirely so, in very great part in this manner only. In the deaf and dumb, of course, the auditory word-centre is never developed, and words are revived only as visual impressions through the visual word-centre.

The position of these centres in the brain is considered by

Dr. Ferrier to be in the case of the visual in the angular gyrus, and in that of the auditory in the upper temporal convolution.

The centres may be damaged differently, so as to result in at least two kinds of defective action: (1) they may be paralysed, or (2) they may take on inco-ordinate action, *i.e.*, supply a wrong word instead of the right one.

I will now consider the modes in which we can (in accordance with a plan which I have learned from Dr. Bastian's lectures) test the efficiency of each centre, and apply them as I proceed to the case in question.

### I. *Auditory Word-Centre.*

(1.) Is the patient's hearing good? This tests the condition of the sensory in-going fibres and of that part of the centre concerned with them. In this case the patient can hear perfectly well.

(2.) Can he understand speech? He comprehends everything that is said to him readily and well.

(3) Can he speak? This tests the outgoing fibres and the portion of the centre connected therewith. But speech may be of three kinds.

(*a.*) Spontaneous: *i.e.*, can the patient express his thoughts as he wishes? Here, he can speak fluently and well, but cannot name objects (except a few familiar ones, and some which he has learnt since his attack). When asked the name of an object, he makes attempts to say the word but cannot do so, and is quite conscious of his failure (paralytic amnesia). Sometimes, however, he says a word somewhat resembling the correct one in sound, as "spinnacles" and "stectacles" for "spectacles," or "rescern" and "concern" for "discern." At other times he will use a word, the meaning of which has a distant connection with the one required, as "mother" for "wife," and "brother" for "son"; or, again, he will use a periphrasis, as, when asked to name a camel, he said, "Egypt—a long way—go a long way—carry things—hot place." All the time he is quite clear as to the word he wants, and will recognise it immediately it is spoken, and will very accurately pick it out from a list of words resembling it in sound, or even from a list of synonyms

Adjectives, verbs, etc., seem to present little or no difficulty, it being almost entirely noun-substantives that are unable to be recalled. Since the patient has been under observation he has learnt many names, and can recall others which formerly he could not do, and so has a fair vocabulary; but still he is constantly at a loss for a word in conversation.

(*b.*) Associational or mnemonic speech; or has the patient the power of repeating things from memory? This one has. He can count well, can say the alphabet, and can repeat the Lord's Prayer. In this case the bond of association or memory is so strong that it lasts uninjured after voluntary speech has been damaged, for the patient can say words under this influence that he cannot say voluntarily.

(*c.*) Imitative speech; or can the patient repeat words after you? This man at first could not repeat anything, but now he can repeat almost any simple word, with only an occasional difficulty. A very long word or a phrase he generally stumbles over more or less.

### II. *Visual Word-Centre.*

(1.) Is the patient's sight good? If so, the in-going fibres are clearly intact; and in this case they are, as the patient can see perfectly well.

(2.) Does he comprehend printed or written words? He does very readily. He reads to himself very often, and obviously understands what he reads, since he can answer questions upon what he has been reading.

(3.) Does he recognise common objects? This man will recognise anything; can describe it, and knows all about it; but cannot name it.

(4.) Can the patient write? This tests the condition of the out-going fibres, and of the portion of the centre connected with them. Here, as in speaking, we have three varieties.

(*a.*) Can the patient write spontaneously? If so, the centre will respond to volitional impulses. In this case there is some defect, for although he can write simple sentences, he cannot write long or difficult ones, such as a description of an object.

(*b.*) Can the patient write things from memory? This is associational or mnemonic writing. Here he cannot very

well. He can write his name, but he has marked difficulty in writing the alphabet, and cannot write the Lord's Prayer, which he can repeat fluently; thus showing that here the defect is in the visual word-centre itself, and not that it is prevented from acting by a defect in the auditory word-centre.

(*c.*) Can the patient copy words—imitative writing? He can do so very accurately, copying either printing or writing, or transferring one to the other. He can also copy easy drawings exceedingly well, considering his abilities.

### III. *Auditory and Visual Word-Centres Combined.*

We have two ways of testing whether these centres can act in unison, and therefore whether their commissural fibres are intact.

(1.) Can the patient read aloud? In doing this the visual word-centre receives a stimulus along its sensory in-going fibres, and passes it along the commissure to the auditory word-centre, where it calls up the corresponding sound, the stimulus to utter which passes along the out-going fibres to the kinæsthetic centre, and finally to the muscular nerves. This man *can read aloud with perfect ease, reading long and difficult words clearly and distinctly.* The fact that he can read aloud and yet cannot name objects shows that there must be a visual *word*-centre, as the auditory word-centre can be stimulated by the reception of impulses from it, but cannot be roused into activity by volitional impulses originated on seeing the object.

(2.) Can the patient write from dictation? This process is the reverse of the preceding, the sensory stimulus reaching the auditory word-centre, and going thence to the visual word-centre along commissural fibres belonging probably to another and a distinct set. In this case the patient has considerable difficulty in writing from dictation, only being able to write simple words, and not always these. Sometimes I have noticed that he was able to write a word spontaneously which he could not write from dictation, but this was not a constant or uniform condition.

We are now met with this problem: If he can read aloud, the commissural fibres must be intact; and if he cannot write

from dictation, they must be damaged. How can we reconcile these contrary propositions?

There are two explanations available:—

I. That there is a double set of commissural fibres—one the audito-visual, and the other the visuo-auditory; and, further, that one of these can be damaged and the other left intact.[1]

II. That after some time the impulses from the visual word-centre, instead of going to the auditory and then to its related kinæsthetic centre, go direct to this latter kinæsthetic centre, passing by the auditory centre. But if the visuo-auditory fibres can be superseded like this, why should not the audito-visual be similarly superseded? In reply we can only make this suggestion, that the auditory word-centre having so much more to do than the visual, it would be an obvious advantage if it could be relieved from transmitting these commissural impulses by their going direct to the kinæsthetic centre. It seems possible that in some people this may be brought about by education and practice, though in the case of a man in the position of this patient, such an explanation would seem hardly likely to be the correct one.

To sum up, then, we find that in this case there is—

I. Some aphasia, though this is much less than it was.

II. Some paralytic amnesia, viz.,—

(*a*.) As regards the auditory word-centre:—

1. Diminished excitability to volitional impulses (spontaneous speech).
2. Normal excitability to associational impulses (reading aloud).
3. Diminished excitability to sensory impulses (imitative speech).

(*b*.) As regards the visual word-centre:—

1. Diminished excitability to volitional impulses (spontaneous writing).
2. Diminished excitability to associational impulses (writing from dictation).
3. Normal excitability to sensory impulses (imitative writing).

[1] On this subject see Dr. Bastian's 'Brain as an Organ of Mind,' 3rd ed. p.640.

III. Some inco-ordinate amnesia extending to the same parts as are affected by the paralytic forms.

Therefore the lesion has damaged the auditory word-centre and the audito-visual commissural fibres, and to a less extent the visual word-centre.

As te the nature of the lesion, I should think that it was probably a thrombosis affecting a small area in the region of these centres as described above.

*Note, October*, 1885.—It is now more than eighteen months since the attack, and the patient is in very much the same condition. He has learnt some more words, and has a little more power in recovering names, but only after considerable delay and many failures. His general health is very good; his grip is, right 85, left 80; and there remains only a slight trace of the right facial paralysis.

# ON A CASE OF BILATERAL DEGENERATION IN THE SPINAL CORD, FIFTY-TWO DAYS AFTER HÆMORRHAGE IN THE CEREBRAL HEMISPHERE.

BY W. B. HADDEN, M.D., AND C. S. SHERRINGTON, M.B.

EDWARD S., aged 63, an upholsterer, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on March 5th, 1883.

The history was that he was found on the top of a tramcar, helpless, but not unconscious. He was at once taken to the hospital. Nothing more was ascertained about him, except that he had been under treatment at an ophthalmic hospital.

*State on Admission.*—Patient is conscious, and quite sensible. He cannot articulate, except to say "Yes" or "No" in answer to questions. He tries to speak, and can make sounds, but is unintelligible. He shuts his eyes when asked, and tries to put out his tongue. He can only protrude it very slightly, and it deviates to the right.

The mouth is drawn to the left. The right arm, which is rigid, is quite powerless, and sensation is impaired. There is also partial loss of motion and sensation of the right leg, but it is not rigid. The knee-jerk is brisk on the right side, and ankle-clonus is present. There is a slight elbow-jerk, but no wrist-jerk on the right side. The plantar reflexes are equal on both sides.

The lungs are emphysematous, and signs of bronchitis audible, mainly at the right base posteriorly.

The heart appears normal. The arteries are tortuous and thickened. There is no albumen in the urine. Ice was applied to the head, and four leeches to each temple. The bowels were freely acted on by croton oil.

*March 9th*.—There is now no aphasia; but there is some difficulty in articulation, on account of the facial paralysis.

The tongue is protruded markedly to the right. The pupils are small, but equal. The bladder is distended, and he passes his water under him.

*April 16th.*—There is no return of power in the arm, and very little in the leg. The right hand is swollen, the skin over the backs of the fingers smooth and devoid of wrinkles, and the nails are more curved over the tips of the fingers than on the left side. There is some atrophy of the muscles of the thumb and of the extensors of the forearm. The elbow and knee-joints on the right side are stiff, and there is pain on making passive extension. The tendon-reflexes of both legs are brisk, but much more so on the right side. There is marked ankle-clonus on the right, but none on the left side.

The patient gradually got more and more drowsy, and albumen was found in the urine.

He died on April 26th, fifty-two days after the onset of the attack.

The temperature of the axilla on the paralysed side was sometimes half a degree or even a degree higher than on the left side, but occasionally it was equal on both sides, and rarely the temperature on the paralysed side was lowered. During the last nine days of life there was marked pyrexia.

*Post-mortem Examination.*—Body of an emaciated old man. Numerous Pacchionian bodies at the vertex of the brain on each side of the longitudinal fissure. The left hemisphere appears smaller and more flattened externally than the right.

The vessels at the base are gaping, and their walls are much thickened. There is no obstruction. There is moderate excess of the sub-arachnoid fluid.

Outside the forepart of the left lenticular nucleus some ochre-coloured clot is seen. Farther back, the clot is larger, the adjoining edge of the lenticular nucleus is lacerated, and the internal capsule between the caudate and lenticular nuclei is involved. The external capsule and claustrum cannot be distinguished. The hæmorrhage also involves the most anterior part of the lenticulo-optic segment of the internal capsule. Except for the laceration of the outer part of the lenticular nucleus, as mentioned above, the corpus striatum and optic thalamus are unaffected. The hæmorrhage measures

about an inch and a half, or rather more, in length, and involves nearly all the motor part of the internal capsule. The white matter forming the roof of the left lateral ventricle is softened and tinged yellow. The cortex everywhere is healthy. No signs of any other lesion in the brain.

There is no evident change in the crus and pons, but the left anterior bulbar pyramid has a slightly pinkish translucent appearance, in marked contrast with that on the opposite side. The degeneration is also visible throughout the entire length of the right lateral column in the cord. It is most marked in the cervical region, less evident in the dorsal; but in the lumbar portion it again becomes very distinct, and reaches up to the periphery of the cord.

There is no difference between the median nerves on the two sides. The lungs are emphysematous, the right side of the heart dilated and hypertrophied, the liver fatty, and the kidneys small and very granular.

In sections prepared from the hardened cord and pons, the following condition is observable.

*Pons and Medulla Oblongata.*—The pyramid of the left side gives a decidedly larger area on cross-section than does that of the right side. There is no obvious difference as regards depth of stain between the right and left sides, either in preparations coloured by carmine, or after Weigert's hæmatoxylin method. Under the microscope the left pyramid proves to be very much degenerated; the right pyramid appears to be undegenerated, save in a small portion of it, lying ventrally and medially, adjoining the grey matter of the "nucleus of the arciform fibres" (Schwalbe). The area given by the degenerating tracts at the level of the lower ends of the inferior olivary bodies is indicated in Fig. 1. The depth of the stippling is intended to be proportional to the degree of degeneration. It is notable that the degenerated area approaches the olive very closely, and that upon the left side three small islets of degenerated fibres lie dorsal to the main pyramidal mass, within the formatio reticularis.

*Spinal Cord*—The right lateral column is slightly smaller than the left, its smallness being due to diminution of its posterior two-thirds, the bold outline of that part on the left,

is on the right replaced by a flatter line. Fig. 2 represents the state of things opposite the fifth cervical nerve-root. Here, in sections stained with logwood, or with carmine or picrocarmine the area of the right crossed pyramidal tract is coloured a little more deeply than normal. This is more obvious in carmine than in logwood preparations, and is most obvious in that part of the tract which borders the direct cerebellar band. No change, however, is obvious in either direct pyramidal tract, or in the left lateral column. In sections stained after Weigert's hæmatoxylin method[1] a degenerative change is at once obvious to the naked eye in both lateral columns and on both sides of the anterior median fissure. On the right side the area of the crossed pyramidal tract is tinged with brown, contrasting with the inky tint of the rest of the antero-lateral column. The region of the direct pyramidal tract also is brownish, and not merely to left of the anterior fissure but to right of it. Further, on the left side the area of the pyramidal tract in the lateral column is brown—though not so strikingly so as on the right. Lastly, the periphery of the antero-lateral column from lip of anterior median fissure round to some millimeters behind the anterior nerve-roots offers a brownish zone, a half or more of a millimeter deep in the section—the method even in sections from well-hardened cord causes much shrinking.

The degree of change in the right half of the cervical cord is greater in the lateral column than in the anterior; in the left half it is greater in the anterior column than in the lateral.

Both in carmine-stained sections, and in those stained by Weigert's method, close examination shows the finer anatomy of the degeneration to be as follows:

In the right crossed pyramidal tract there is some excess of connective tissue, especially of large deeply-staining cells which lie between the cross-sections of nerve-fibres. Individual nerve-fibres are not so distinct and easily countable as normal, although many are perfectly distinct. Many of the stromal spaces are vacant; presumably because the cross-sections of the fibres have dropped out in the process of preparing the specimen for the microscope. In some of the stromal spaces

[1] 'Fortschr. der Med.,' April 15, 1885.

lie large coarsely granular, rather highly refracting, and ill-stained, rounded bodies (cf. Fig. 7 *d*). They are not deeply coloured by carmine or Weigert's hæmatoxylin method. A nucleus is not certainly evident in them, but with carmine one part of them often colours more deeply than the rest. Whether they are similar to the "granulation corpuscles" discussed and figured by Bastian,[1] or whether they are portions of much altered nerve-fibres, is not clear to us. Some stromal spaces are filled by ill-defined palely-staining homogeneous masses; some contain simply an almost uncoloured granular *débris*; occasionally are seen cross-sections of fibres to all appearance healthy, but that the axis-cylinder stains more deeply than normally and appears enlarged. Without more exhaustive examination, the impression left upon us is that the fibres degenerating, although not of the largest of the antero-lateral column, are of the larger rather than of the smaller—but are not all of equal calibre.

Similar changes are present in the left lateral column, but there the increase of connective-tissue elements is very slight. In the anterior columns, as in the left lateral, the changes are almost confined to the nerve-fibres, the stroma not being obviously thickened. In the dorsal cord, Fig. 3, at the level of the second nerve-root, the bilateral character of the degeneration is evident. The change is still most marked in the right lateral and left anterior columns; the shape of the area involved is not much different from that of the cervical region.

Fig. 4 shows the change at the level of the 10th dorsal nerve, here again evidently bilateral; but of somewhat altered shape. The area of the direct cerebellar tract has been encroached upon, and as the cerebellar fibres are here scattered, and are set in much tissue continuous with the deeper pial layer, a little care is wanted not to suppose that the degeneration-field here extends to the periphery of the lateral columns from the posterior root forwards. At this level no degenerating fibres are observable at the right border of the anterior fissure.

In Fig. 5, opposite the 2nd lumbar nerve, it is seen that the

[1] 'Trans. of Medico-Chir. Soc.,' vol. l. p. 525. Pl. XI. Fig. 20.

degeneration area lies further outward from the central grey matter. Degenerating nerve-fibres still are present in the left border of the anterior fissure.

In Fig. 6, at the 1st sacral root, the degeneration is still bilateral in character, but absolutely confined to the periphery of the lateral columns, as a triangular area, more obvious on the right side.

The median nerve of the paralysed right limb was healthy.

The above case is interesting, from the opportunity offered for microscopic examination of the secondary degeneration in the cord, so early as seven weeks after the occurrence of the cerebral lesion. That the degeneration in the cord was due to the hæmorrhage which occurred fifty-two days before death, there is no good reason to doubt. It is noteworthy that the degeneration in this early stage shows very little sclerosis; even in the crossed pyramidal tract in the right lateral columns where it is most, it is quite slight. It has been shown that in the dog the amount of sclerosis in the degeneration patch is proportional to the age of the degeneration, *i.e.* the farther the degeneration dates back, the more the fibrous tissue in the track of degeneration, until a period is reached, within the year in the dog, at which the only evidence of a degeneration-process that has occurred is the scar it has left behind. By this criterion a glance at a carmine-stained section of the cord from the present case would show that death had followed at a comparatively short period the initial lesion in the brain.

The process of degeneration, in the present case, seems as far advanced in the lumbar region as in the cervical. This with the early date is interesting, and agrees with observations to the same effect in the dog.[1] In fact the name "descending" degeneration given to the secondary degeneration of nerve-fibres distal to the seat of initial lesion seems scarcely justified. It implies an idea of change observably extending downward along the fibres, whereas there is great dearth of observation of such a progression. *A priori* we conceive there is as much reason to suppose the change an ascending one, or one occurring simultaneously throughout the length of a fibre, as to imagine it descending. Of the fibres in the spinal cord

[1] Sherrington, 'Journ. of Physiol.,' vol. vi. p. 177.

trophically dependent on the brain, the earliest portions to suffer after brain injury might well be supposed those at greatest distance from their trophic centre. Were speculation useful here, many arguments suggest themselves for and against each of the three views. But we would remark that serious objection against the name "descending" degeneration can be urged, until more observation of its descent are forthcoming.[1]

The Figures 1 and 2, to which references will be found on p. 511, show a point on which we would lay stress; the evidently large diminution which the degenerating tract undergoes in amount as it passes from the medulla oblongata into the spinal cord, in the region of pyramidal decussation. Into what structures do the manifold degenerated fibres stopping short at this level embouch?

A point of great interest in the present case is that a degeneration doubly bilateral in the spinal cord has resulted from injury confined to one hemisphere of the brain. Pitres has described such cases.[2] In the dog analogous cases have been observed by Moeli,[3] by Löwenthal,[4] and by one of us.[5] Schäfer[6] also, as long ago as 1883, noticed that in the spinal cord of a monkey operated on by Drs. Ferrier and Yeo, the cervical enlargement contained a degeneration in the lateral column on the same side as the cerebral injury.

In the present case, the degeneration in the lateral column on the same side as the hemisphere injured, when compared with that of the crossed pyramidal tract opposite, presents the same characters as in the dog; it is more diffused, less defined, with edges softer. Although the difference between the degenerations on each side of the anterior fissure is not great, still differences the same in kind are present there also, the right-hand degeneration being more diffuse. In the dog, one of us was led to believe that the degeneration on the same side as the cerebral injury either appeared rather later, or, if it began simultaneously with that of the crossed tract, was at first quite slight, and only later, when that of the crossed tract had

[1] The only support we know of for the term is by Schiefferdecker. Virchow's 'Archiv,' 67.

[2] 'Archives de Physiologie norm. et path.,' vol. v. 3rd series.

[3] 'Archiv. f. Psych.,' vol. x.

[4] 'Zeitschrift Zoologie,' Geneva, 1885.

[5] 'Jour. of Physiol.,' vol. vi.

[6] 'Jour. of Physiol.,' vol. iv.

already been a little time in full play, reached its own stage of full activity. If one may from amount of sclerosis present judge the age of a degenerative lesion, one would say that in the present instance the lesion in the right lateral column had been in existence longer than that in the left. In the dog too, especially when the degenerative lesion is small, it seems that the degeneration on the same side as the cerebral injury does not progressively diminish in quantity from above downward, as does the lesion of the crossed pyramidal tract. In the case before us, the left-hand degeneration does not at any lower point in the spinal cord seem larger than at some point more proximal. The only hint of such a character is in the comparison of sections from the levels of 3rd and 5th cervical nerves respectively; the degeneration in the left lateral column may be slightly more extensive at the 5th than the 3rd.

Pitres does not find any degeneration in the pyramid of the side opposite the cerebral lesion. It must be remembered that Pitres is always dealing with the scar left by the degeneration rather than with the degeneration itself. His earliest case is one of more than six months' standing. It seems doubtful whether the degeneration of a small number of fibres scattered somewhat sparsely through a tract of uninjured fibres would leave any scar—at least, any scar detectable by present methods of observation. In the present case, as above stated, we believe the degeneration to be bilateral in the medulla oblongata although extremely asymmetrical there—much more asymmetrical than at any point in the spinal cord below the decussation of the pyramids.

There are some points of clinical interest in this case to which we would briefly call attention. The rigidity of the paralysed limbs occurred at the onset of the attack, and persisted until death. It is usually asserted that early rigidity in hemiplegia is due to irritation of the pyramidal-tract fibres, and this assumption receives some support from the present case. Whatever may be the explanation, there is no doubt that the so-called early rigidity in some instances remains permanent, no interval elapsing between it and the slow contracture which results from secondary spinal degeneration. We have for some time had a case under observation, in which

rigidity of the paralysed arm and leg ensued immediately on the attack. A year later the rigidity was still present in a very marked degree.

The early occurrence of ankle-clonus in our case deserves some notice. This we have observed in other cases in which no obvious rigidity was present. Attention has also been directed to this subject by Pitres.[1]

Although, in the case we have narrated, the secondary degeneration was bilateral, it is interesting to note that rigidity and ankle-clonus were not present in the non-paralysed side. The only indication of any organic change was an exaggeration of the knee-jerk on that side.

On this point, Pitres, in the paper to which we have already alluded,[2] says: "Bilateral exaggeration of the knee-jerk and bilateral contracture of the lower limbs may exist in cases in which the secondary sclerosis is perfectly unilateral; and, on the other hand, they may not be present when the secondary sclerosis occupies both lateral columns."

A similar opinion had already been expressed by one of us on theoretical grounds.[3]

It was then suggested that the occurrence of ankle-clonus and rigidity on the non-paralysed side in hemiplegia might sometimes depend on irritation conveyed from the motor cells on the hemiplegic side to those on the opposite side of the spinal cord. The intimate connection between the nerve-nuclei which preside over the lower limbs, and the comparative independence of the motor nerve-cells controlling the movements of the upper limbs, explain many facts of the highest clinical interest.

[1] 'Brain,' 1884, p. 310.
[2] 'Archives de la Physiologie norm. et path.' 15 Février 1884.
[3] Hadden, in 'St. Thomas's Hospital Reports,' 1882.

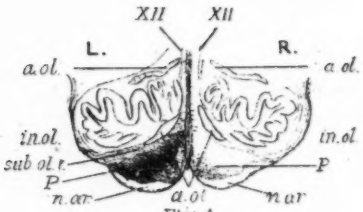

Fig.1

Fig.2

Fig.3

Fig.4

Fig.5

Fig.6

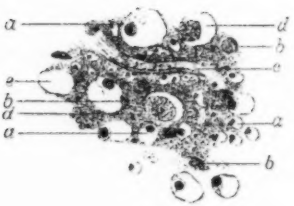

Fig.7

C.S.S.dd. Danielsson & Co. lith.

## EXPLANATION OF PLATE.

Abbreviations :—

| | | |
|---|---|---|
| in. ol. | .. | inferior olivary body. |
| P. .. | .. | Pyramidal tract. |
| sub. ol. t. | .. | subolivary tract (olivenzwische schicht). |
| a. ol. | .. | accessory olivary bodies. |
| n. ar. | .. | nucleus arciformis. |
| R. .. | .. | right-hand side. |
| L. .. | .. | left-hand side. |

Fig. 1. Cross-section through medulla oblongata, opposite the lower ends of the inferior olivary bodies.

„ 2. Ditto, through the spinal cord opposite the 5th cervical nerve root.

„ 3. Ditto, ditto, 2nd dorsal nerve.

„ 4. Ditto, ditto, 10th dorsal nerve.

„ 5. Ditto, ditto, 2nd lumbar nerve.

„ 6. Ditto, ditto, 1st sacral nerve.

„ 7. Portion of the region of the right crossed pyramidal tract at the level of the 10th dorsal nerve, at the spot marked * in Figure 4. Zeiss—Objective F. Ocular 2, drawn with camera. Preparation coloured by Beale's carmine-fluid ; mounted in Canada balsam.

*a*, normal nerve-fibres seen in cross-section.

*b*, nuclei of neuroglia cells.

*c*, capillary blood-vessel.

*d*, stromal space in which the nerve-fibre has degenerated—occupied partly by a coarsely granular, palely staining, non-nucleate mass.

*e*, stromal space empty, perhaps because its nerve-fibre was degenerated.

All the figures except the last are magnified two diameters. In the lithographic reproduction, especially of Figs. 2, 3, 4, 5, the stippling, indicating the areas of degeneration, is much darker than in the original drawings.

# THE INFLUENCE OF THE INTENSITY OF THE STIMULUS ON THE LENGTH OF THE REACTION TIME.

BY JAMES McKEEN CATTELL,

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DURING the past two years, Dr. G. O. Berger and I have been carrying on a series of experiments in the Psychological Laboratory of the University of Leipsic, looking to determine the relation between the intensity of the stimulus and the length of the reaction time. Wundt, Exner, and others had already made experiments on this subject, but it seemed to need a more thorough investigation.[1] We undertook to determine the influence of various intensities of the electric shock, and of light on the length of the simple reaction time, and on the reaction time complicated by the addition of simple cerebral operations.

The term "reaction time" is now generally understood. If one lifts one's hand as soon as possible after the sudden appearance of a light, the interval between the application of the stimulus and the beginning of the muscular contraction is a reaction time. In order to investigate the influence of various intensities of light on the length of this time, we used a light produced in a Geissler's tube by an induction current from six Daniell cells. This light we took as normal, and kept constant. We then arranged five weaker intensities by putting smoked glass before the light. The amount of light transmitted through the smoked glass we determined photometrically. If we set the intensity of the normal light VI = 1000, then the intensities of the lights would be

| I. | II. | III. | IV. | V. | VI. |
|---|---|---|---|---|---|
| 1 | 7 | 23 | 123 | 315 | 1000 |

We further obtained two still brighter lights (vii and viii) by

[1] Wundt, 'Physiologische Psychologie,' 2. xvi.; Exner, 'Hermann's Physiologie,' 2. 2. iv.; Kries u. Auerbach, 'Archiv f. Anat. u. Physiol.,' 1877; Vintschgau u. Hönigschmied, 'Pflüger's Archiv,' xii.; Wittisch, 'Zeitschr. f. Rat. Med.,' xxxi.

means of lenses, but could not determine with our photometer the relation of these to the normal intensity. The observer sat in the dark, and looked through a telescopic tube at the point where the light was to appear. The following table gives the average of 150 reactions made by each of us with the several intensities. No reactions at all were omitted in taking the average. The second line, marked M, gives the average of the variation of each from the average of all the reactions; that is, if A is the average of $n$ reactions $a$, $b$, $c$, $d$, then

$$M = \frac{(A-a)+(A-b)+(A-c)\ldots}{n}$$

all the differences being taken as positive. M shows us how much the reactions differ from one another, and when we know the number of reactions, we can find the probable error of the average. In the table ·001s. is taken as the unit of time.[1]

TABLE I.

| | B. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Intensity. | I. | II. | III. | IV. | V. | VI. | VII. | VIII. | Average. |
| Time. | 308 | 235 | 208 | 200 | 192 | 195 | 177 | 168 | 210 |
| M. | 26 | 18 | 16 | 15 | 15 | 17 | 18 | 16 | 18 |

| | C. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Time. | 251 | 175 | 160 | 148 | 147 | 143 | 135 | 128 | 161 |
| M. | 30 | 17 | 16 | 14 | 15 | 13 | 16 | 19 | 18 |

It will be seen from the table that when the light is taken very weak, just strong enough to be seen, the times are the longest and (with one accidental exception, B between V and VI) the greater the intensity of the light, the shorter the time of the reaction. I cannot, however, formulate a general law from the table.

In substantially the same manner the relation between the

[1] It should be mentioned that in our first experiments the absolute time is not certain. The times were measured with aid of an electro-magnet, and we assumed that when a current was sent through the coil, the armature was attracted instantaneously. This is not the case, and I have made an allowance for the error, but the times may be as much as ·01s. wrong. At all events the relative times are correct, as the error was constant.

strength of an electric shock and the length of the reaction time was determined. The shock was received on the left forearm, and the reaction made with the right hand. We used four intensities; the strongest, IV, somewhat painful, the weakest, I. just enough to be felt. The two intermediate intensities made up, as far as we could judge, four equal steps. The averages of 150 reactions by each observer and on each intensity are given in the table.

TABLE II.

| Intensity. | I. | II. | III. | IV. | Average. |
|---|---|---|---|---|---|
| | | | B. | | |
| Time. | 182 | 163 | 158 | 160 | 166 |
| M. | 17 | 14 | 12 | 11 | 13 |
| | | | C. | | |
| Time. | 164 | 155 | 132 | 131 | 145 |
| M | 19 | 18 | 14 | 14 | 16 |

It will be noticed that with the electric shock, as with light, the time of the reaction becomes shorter as the stimulus becomes stronger. The differences are not, however, so great, and for the intensities III and IV, the times are about the same; with IV the reaction was probably retarded, because the shock was painful.

In connection with the experiments on the intensity of the light, we made others to determine whether or not the quality of the stimulus, that is, the colour of the light, has any influence on the length of the reaction time. The averages of 180 reactions made by each of the observers, and on each of the six colours used, are given in the table.

TABLE III.

| | White. | Red. | Yellow. | Green. | Blue | Violet. | Average. |
|---|---|---|---|---|---|---|---|
| B. | 196 | 203 | 192 | 196 | 199 | 201 | 198 |
| C. | 155 | 162 | 160 | 156 | 161 | 153 | 158 |

The table does not show any decided difference in the times for the several colours. Violet and green, which I have found (see last number of 'BRAIN') must work longer on the retina than the other colours, in order that a sensation may be called forth, do not seem to cause a longer reaction time; this is because the reaction is made on the light, without waiting until the colour has been distinguished.

The time is longer when it is necessary to distinguish the colours before the reaction is made. We can determine this time, if instead of always reacting as quickly as possible we use two lights of different colours, say blue and red, and let the subject react only on one of them. The subject does not know which light is to come, but is to lift his hand as quickly as possible if it is red, but not at all if it is blue. We thus add to the simple reaction the time it takes to see whether the light is blue or red, and complicate somewhat the process of volition in the simple reaction time. We can further let the subject lift his right hand if the light is red, his left hand if it is blue; we then have, besides the time necessary for the simple reaction and for distinguishing the colour, the time it takes to make a choice between two motions. The results of experiments made with three intensities of light (V, III, and I) are given in the table.

TABLE IV.

| | B. | | | C. | | |
|---|---|---|---|---|---|---|
| | V. | III. | I. | V. | III. | I. |
| Reaction time | 189 | 218 | 273 | 189 | 209 | 303 |
| Reaction with Perception time | 238 | 293 | 373 | 274 | 328 | 417 |
| Reaction with Perception and Will time | 287 | 320 | 393 | 356 | 388 | 495 |
| Perception time | 49 | 75 | 100 | 85 | 119 | 114 |
| Will time | 49 | 27 | 20 | 82 | 60 | 78 |

It seems from the table that the time it takes to see or perceive a colour becomes shorter as the intensity of the light becomes stronger, but that the will time is not a function of the intensity of the stimulus.

## Clinical Cases.

# A CASE OF AMYOTROPHIC LATERAL SCLEROSIS WITH CLONUS OF THE LOWER JAW.

BY C. E. BEEVOR, M.D., M.R.C.P.,

*With a* NOTE *on the* JAW-JERK, *or* MASSETERIC TENDON REACTION, *in* HEALTH AND DISEASE.

BY A. DE WATTEVILLE.

As Dr. Gowers has called my attention to a case published in America, in which an increased tendon-reflex of the muscles of the lower jaw was obtained, I have thought that this case, which I saw four years ago when resident at the National Hospital for the Paralysed and Epileptic, might be of interest.

The case was under the care of Dr. Ramskill, whom I have to thank for allowing me to publish it.

The patient, a woman aged 46, with no previous or hereditary history of importance, noticed one morning, nine months before admission, that the left side of her mouth felt "dead"; six weeks later the left arm, one morning, felt numb, and she could not hold objects with it; the forearm gradually got worse, and has been paralysed for five months; symptoms of bulbar paralysis gradually came on; in four months from onset her speech became affected, and in eight months swallowing was difficult. Seven months from onset, the right hand began to be weak.

On admission, she had all the signs of bulbar paralysis: difficulty in showing the teeth; inability to swallow, with regurgitation of fluid through the nares; inaction of the soft palate; speech unintelligible, only being able to make a moaning sound; her tongue she could not protrude past the teeth, although it was not much wasted; the masseters and temporal muscles were very weak.

In the left upper limb the small muscles of the hand, and the forearm were much wasted, and the wrist and fingers quite powerless; there was less wasting of the upper arm and shoulder, and very slight movement was possible in the left elbow and shoulder-joints.

In the right arm the ball of the thumb and interossei were

somewhat wasted, and the movements of the whole arm were weak.

In the left arm there was considerable rigidity of the joints, and this was present to a less degree in the right arm.

With regard to the lower limbs, they were not wasted; she could walk well, and the only thing remarked was some rigidity about the joints.

The *knee-jerks* were excessive on both sides, perhaps the left more than the right; but there was *no ankle-clonus*.

On filliping the lower end of the radius of either side, a very active contraction of the flexors of the elbow was produced.

The symptom which I think the most interesting in this case is the *clonus of the lower jaw*; this could very readily be produced by placing the finger on the teeth of the lower jaw and then depressing it, when immediately the muscles which closed the jaws passed into a state of clonic contraction, and the lower jaw vibrated as long as the pressure was kept up by the finger on the teeth; in fact, it behaved in the same manner as the heel does when ankle-clonus is produced. Clonus could also readily be produced by striking the masseters; and also if the zygomaticus major was percussed, retraction of the corner of the mouth ensued.

I do not know whether a similar case has been observed, but I do not remember to have heard of any one having been reported.

It is interesting to notice that the sclerosis of the cord was confined chiefly—if not almost entirely—to the medulla and upper part of the spinal cord, so that clonus of the lower jaw and radius tap-contractions were very readily obtained, while, on the other hand, ankle-clonus, which was tried for on several occasions, was absent, and though contraction of the extensors of the knees could be obtained by pressing down the patella from above, this method failed to produce clonus of these muscles.

According to the patient's account, the loss of power in the left arm occurred suddenly; but whether this was really the case, it is difficult to decide.

Since seeing this case, it has frequently occurred to me whether the chattering of the teeth produced by cold or by a rigor could be merely a clonus of the muscles of the jaw, and whether in these conditions the "tendon-reflexes" of all the muscles of the body are increased; as this is only thrown out as a speculation, I shall certainly test the tendon-reflexes in any case of rigor I may happen to see. From my personal experience, I think that the knee-jerk is increased after a prolonged stay in a cold bath; but I have not as yet been able

to produce ankle-clonus, though I have heard on good authority of its occurrence in a healthy individual under such conditions.

I may add that the patient soon returned home, and I lost sight of her, and I am unable to give details of her further progress.

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## NOTE.

As it does not appear to be generally known that a "jaw-jerk" can be readily elicited by an appropriate stimulus in most healthy persons, I take the opportunity offered by the publication of Dr. Beevor's observation to call the attention of neurologists to this point. The phenomenon is clearly of the same nature as that of the "knee-jerk," and is due to the sudden stretching of the masseter and other muscles of mastication. Hence the name I have ventured to give to it, in preference to the longer and less accurate term mandibular (or masseteric) tendon-reaction (or reflex).

The method of producing the contraction, which I find most effectual and convenient, is to introduce into the experimentee's opened mouth a tongue depressor or a paper knife, which is made to press with its flat surface upon the teeth of the lower jaw. It is better to place it somewhat laterally upon the canine and premolars than in the middle line upon the incisors. The blade is held down with a firm pressure, care being taken that the jaw is not fixed by any undue muscular contraction; the experimentee should just resist the strain downwards, and nothing more. A sharp tap with a convenient object, such as a ruler or a thin-bound volume, but best of all with a percussion hammer, is then struck upon the paper-knife, close to the teeth. A contraction of the stretched muscles then occurs of more or less amplitude, but which is usually quite appreciable to a finger introduced between the molar teeth on the opposite side.

In many cases, especially in such where one finds the usual tendon-reactions exaggerated, one readily obtains a very lively jaw-jerk. This fact, I feel bound to state, was quite familiar to me before it came to my knowledge that the phenomenon had been observed in a case in America. I have not been able to ascertain the author of, any details about, nor the reference to, this observation.* But it is to Dr. Beevor's mention of the occurrence of jaw-clonus in the case he now publishes that I owe to have had my attention drawn to the mandibular tendon-reactions.

When exaggerated, the jaw-jerk may be elicited by using the fingers to depress and mediately percuss the lower jaw. The left forefinger is placed either upon the teeth, according to the plan already described, or outside upon the projecting mental ridge, and the right index and middle finger used instead of the hammer.

* Both Dr. Gower's and Dr. Beevor's information concerning this case is limited to the bare fact of its existence.

Or, again, the hammer may be made to strike the jaw directly, on either side of the chin. I have thus, in one way or another, obtained evidence of what appears to be abnormal excitability in the nervo-muscular apparatus of mastication in various conditions, such as organic or functional nervous disorders, in convalescence from acute diseases, &c. Abnormal diminution, or absence, of jaw-jerk, on the other hand, is a subject which I prefer to pass over for the present, and reserve for a future occasion, when my experience of the variations compatible with health is more precise and extensive. It is possible that in time the condition of the jaw-jerk may acquire a diagnostic value in cases of bulbar or other disease. With reference to jaw-clonus (which it is the credit of Dr. Beevor to have been the first to describe in the present article), I may mention that I have, since he told me of it, demonstrated its occurrence, both in purely functional nerve disorder (as in a case of hysterical, tonic, and clonic spasms of the limbs, spontaneous and provoked), and in organic disease (as in a case of right hemiplegia with left hemiparesis, articulatory disturbance and ocular troubles). The methods for eliciting the clonus are the same as those just described for bringing about the simple jerk.

In conclusion, I will state that I have endeavoured, with the kind assistance of my friend, Dr. Waller, to obtain myographic tracings of the jaw-jerk, in order to determine the latent time of the phenomenon. We also attempted to record, on the revolving cylinder, the rhythmical movements which constitute the jaw-clonus. Our results give very nearly ·02 second for the latency of the contraction of the muscles of mastication, when excited by percussion of the jaw according to the method just described. We found this period to be the same, both in the healthy and the diseased subject. Should these measurements be confirmed by further investigations, they will furnish another argument in favour of the view, that the so-called "tendon-reflexes" are phenomena due, not to a true reflex excitation, but to the direct stimulation of the muscles by the sudden extensile impulse of the blow. Dr. Waller, in some recent experiments, has determined the latency of the closure of the eyelid to the stimulus of a strong light. This process, the shortest true reflex known, occupies more than twice as long as the jaw-jerk, viz. about ·05 of a second.

A. DE WATTEVILLE.

# PRIMARY SPASTIC PARALYSIS AND PSEUDO-HYPERTROPHIC PARALYSIS IN DIFFERENT MEMBERS OF THE SAME FAMILY, WITH PROBABLE HEREDITY IN BOTH.

BY R. W. PHILIP, M.A., M.B.

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THE following cases form a family group of considerable interest and importance. For the opportunity of studying them I am indebted to the kindness of Professor Grainger Stewart, under whose charge they have lately been, in the Royal Infirmary, Edinburgh.

The group consists of a father, G. P. sen., and two sons, A. P. and G. P. jun. As the family history is common property, I shall, before describing the individual cases, narrate so much of this as seems important. The father of G. P. sen. died about the age of sixty, of apoplexy, and the mother, aged fifty, from gradually advancing debility. Of this marriage, there were nine children, three daughters and five sons, beside the patient. Of all these the history is good. Uncles and aunts on both sides were numerous, and are reported to have been strong and healthy. G. P. sen. married thirty-two years ago, and his wife is alive and strong, aged fifty-three. The wife's family history is less perfect. A female cousin, aged fifty, has been so completely paralysed, as to be unable to feed herself for eighteen years, while an uncle, aged sixty-six, has been similarly paralysed for a number of years. By this marriage also there were nine children, whose individual history is of interest:—(1) girl, drowned accidentally, aged seven; (2) girl, alive and healthy, aged twenty-eight; (3) boy, died, aged fifteen, apparently of pseudo-hypertrophic paralysis. The father says that "for several years he hadn't the use of himself;" (4) boy, died aged eleven, with symptoms of dropsy; (5) girl, died, aged four, of scarlatina; (6) boy, alive and healthy, aged eighteen; (7) boy, died, aged fifteen, of pseudo-hypertrophic paralysis. His case was de-

scribed as such by Dr. Macphail, Whifflet,[1] (8) Loy, A. P. (*vide infra*); (9) boy, G. P. jun. (*infra*).

Of the three at present in hospital,—

G. P. senior, aged sixty, was admitted on account of inability to walk properly. He has worked as a miner since he was little more than twenty, for the first twenty-five years among ironstone, latterly among coal. His occupation, besides taxing his strength much, involved continued working on his side, in cramped and constrained postures, often partially below water for hours. The space in which he worked not unfrequently measured from 2 feet to 2 feet 10 inches in height. Twelve or thirteen years ago, he met with a severe accident. The guiding rope for the hutches or low waggons, used for carrying coal from the cutting to the bottom of the shaft, broke, and his clothes were entangled by the rapidly passing hutches. He was dragged for about 100 yards, being much squeezed, and sustaining severe injury to the right leg and hip. In consequence of this he was laid aside for twenty-six weeks. On recovering, he kept to coal-mining, as being lighter, though the position necessary for work was more cramped. At this time he began to experience a sensation of stiffness in the right lower limb, which made the bent posture difficult. He also noticed that when he put his foot to the ground, the whole limb began to shake. He occasionally suffered from darting pains, described as "burning," which passed up from the sole of the right foot to the loins. These gradually grew more constant and the general condition worse, till, two years ago, he had to give up work altogether, from inability to walk. In addition to the increased stiffness and pain, his right foot tended to drag and trip easily. He had great difficulty in extending the leg at the knee and hip, and this difficulty made him walk on his toes. The tendency to trembling of the limb grew more marked. A year ago, the left leg showed signs of becoming similarly affected, and during the last two months he has felt a cold, aching pain in the upper arm, during movement. Three days ago he began to experience pains in the right shoulder, and the "startings," previously experienced in the leg, threaten to appear here also. The patient's home surroundings have been comfortable. He has always had good food. He frequently got tipsy on pay-nights. At other times he was sober. There is no history or suspicion of syphilis.

*Present condition.*—Patient is a man of 5 feet 9 inches, of fair development and muscularity. Scattered over his face and extremities are numerous pigmentation spots and cicatrices, the remains of injuries received from time to time in the course

[1] 'Glasgow Medical Journal,' July, 1882.

of his work. The lower part of the thorax projects abnormally, and is separated from the abdomen by a well-marked horizontal depression. A certain degree of lordosis is present.

In standing, he stoops a good deal, and supports himself entirely on the left leg, the right being flexed at the knee and hip, and forcibly adducted, so as to cross its fellow. The left leg is slightly bent at the knee and adducted. Talipes equino-varus is not marked. In walking, he makes use of a stick in his left hand, and in his right, a triangle stool of appearance like , the vertical side being held next himself, so as to afford a broad basis of support on the side towards which he tends to fall. As he lies in bed, the right thigh is flexed and adducted, and the right leg is flexed at the knee, the left to a less extent. The muscles of the right thigh, and the lower part of the abdominal muscles of the same side, show a high degree of contracture, while the calf muscles are flabby. On the left side, the muscles of the thigh, especially the ham-string and the adductors, feel rigid. Regulated movements of the lower limbs are practically abolished. The upper limbs show no lack of power of co-ordinated movement. Ankle-clonus can sometimes be elicited, but the rigid condition of the parts considerably interferes with its demonstration, as also with the knee-phenomenon. Over other tendons, however, a slight tap produces exaggerated movement. This is specially well marked at the elbow. There is no important alteration in the organic reflexes, though the initiation of micturition is delayed. The superficial reflexes are normal. The affected muscles exhibit a diminished reaction to faradic stimulations. The reaction to galvanisation is generally lessened, but the right hamstrings exhibit increased irritability, being also abnormally sensitive to mechanical stimulation. There are no qualitative changes. An aching pain and a sensation of stiffness is experienced in the right knee, thigh, hip and groin, and occasionally in the shoulder and neck towards the same side. Patient also describes a feeling of coldness and numbness which extends from the right foot to the corresponding thigh. Sensibility is unimpaired. The special senses are intact, with the exception of sight, which has been much less acute since the accident twelve years ago. There is no ophthalmoscopic change. Of trophic disturbances, besides the wasting of certain muscles there is to be noted the presence of a number of bald patches—true alopecia areata—scattered over the head, and of erythematous and scaly blotches about the feet. His memory is not good, and he is apt to make contradictory statements, so that the sifting of the history has been matter of difficulty. In other respects, the cerebral functions seem normal. Percussion over the spine reveals the presence

of a fairly well-defined sensitive area extending from the 7th to the 11th dorsal vertebra, and less distinctly as far as the sacrum. The other systems seem healthy.

A. P., aged thirteen, was admitted on account of loss of power in body generally. Up to six years of age, he seems to have been a strong, healthy child. When two years old, he is reported to have fallen from a high bed to the floor on his back, but according to the parents, this accident did not produce permanent injury. He began school at the age of five, but in the course of a year it was observed that he had difficulty in walking. He was easily tired, and tended to fall unaccountably. He was, therefore, removed from school, as the parents feared he might tumble into a canal along which he had to pass. His plump, healthy appearance remained undisturbed, but it was noticed that his gait became awkward, his legs being swung round, and his body swaying from side to side. Complaint was made of pain in the small of the back. The weakness gradually advanced, the tendency to fall increased, the muscles of the trunk and arms becoming affected, so that when down, he had difficulty in raising himself. The parents, who as the history shows, have had ample reason to become observant, state that all this time they were of opinion that a number of the muscles gradually increased in size, while others diminished. Three years ago, the movements at the shoulder joint became much restricted. A year later, his legs absolutely refused to support the weight of his body, and he had to be constantly on a chair, bolstered up with pillows. His left arm was elevated by bending the head forward, taking the finger tips between his teeth, and raising the head again. Within the last year, the back has become much weaker, with a marked convexity posteriorly, and he had to be strapped to a chair. For a number of weeks he has been confined to bed.

*Present condition.*—Patient is a healthy-looking boy, with contented expression. His face and the backs of his hands have a faint brownish tinge, as if sunburnt. His facial expression suggests a constantly full mouth, and the large tongue tends to protrude, but there is no trickling of saliva. The head appears heavy, and inclines forward, but its movements are natural. He has a well-marked goitre. The patient usually lies or half sits in bed, his back supported by pillows, most of his weight resting on the left buttock. The left leg is flexed, and lies on its outer aspect, with the foot and ankle passed underneath the right knee. The right leg is also flexed, and lies on its inner aspect, the knee overlapping the left ankle. The right foot is partially extended, and lies partly on its inner edge, and partly on the inner half of the sole. The pose of the arms suggests

powerlessness, the upper arms lying closely applied to the side of the body, and the forearms resting on the thighs. The lower part of the thorax bulges considerably forwards, and a marked furrow exists between it and the full abdomen. From the third dorsal vertebra to the sacrum, the spinal column is curved backwards and to the right, the curve being greatest in the lowest part of the dorsal region. Patient cannot raise himself sufficiently to attempt standing, but he is still able slowly to bring his right hand to his head by the action of the trapezius and scapular muscles, and the muscles of the forearm attached to the lower end of the humerus. The left arm is quite helpless. Of the muscles, some are markedly increased in bulk, others reduced. The tongue is distinctly enlarged, and rolls about in a heavy, cumbrous fashion. The cheeks are relatively fuller and feel abnormally firm. This seems due to enlargement of the buccinators. The muscles of expression act sluggishly. The muscles forming the floor of the mouth are thickened. The latissimi dorsi are wasted. Both deltoids are full and firm in the lower half, but atrophied in the upper. The trapezius is wasted, especially of the left side. The inferior scapular muscles appear in fair condition. The right pectoral is atrophied, and the power is abolished, the left less so. Biceps is atrophied on both sides, brachialis anticus less so. Neither triceps is much altered. Supinators, pronators, flexors and extensors of forearm and hand are well represented, and power of movement seems natural, though sometimes there is loss of time in its initiation. The muscles of the hand appear intact. In the lower extremities, the calves are remarkably enlarged, and the buttocks to a less extent. Both sets of gluteal muscles are hard and firm, but there is almost total loss of power. The flexors and adductors of the thighs are firm, but almost powerless. The quadriceps extensor is hard and full, especially on the right, but power is much reduced. The hamstrings are more natural in appearance and strength. Both sets of calf muscles are much increased in bulk. There is a slight degree of talipes equino-varus. The muscles on the front of the leg are fuller and rounder than normally. Extension of the foot is possible, but power of flexion is lost. Of the back, the muscles on the right side appear fuller and firmer, those on the left are flabby. Both the atrophied and the hypertrophied muscles react less readily than normally to faradic and galvanic stimulation. No qualitative changes were detected. To the hand, the skin over the enlarged muscles feels considerably warmer than that of the rest of the body, but careful examination with the surface thermometer revealed no appreciable alteration. The organic reflexes seem fairly natural, though for the last few weeks there has been a

tendency to incontinence of urine. Patellar tendon-reflex is abolished, and there is no ankle-clonus. The superficial reflexes are natural. There are no sensory disturbances. There is no tenderness of the spinal cord. The intelligence is good, speech and memory are natural. In other respects, the boy seems in good health. He eats and sleeps well.

G. P., junior, aged eleven, was admitted on account of weakness in legs, and awkwardness of gait. He was in regular attendance at school up to the date of admission. There is no history of accident. Four years ago his parents observed that he walked more stiffly than was natural, and that his knees appoared weak. He used to trip occasionally. Two years later, it was remarked that the left foot was no longer placed flat on the ground in walking, the heel being distinctly raised.[1] This alteration grew more marked, and was followed in three or four months by similar appearances in the right foot. Both feet tended to drag, and stumbling became more frequent. He never complained of pain. He never showed the "waddling" gait of his brother, nor did he tend to fall down "all of a heap." His calves do not appear to the parents to have increased in size.

*Present condition.*—He is a strong-looking boy of 4 feet 9½ inches. Weight, 5st. 5½ lbs. Development and muscularity good. He runs about the ward, apparently with little difficulty. Close examination shows that he walks on his toes, his boots being accordingly altered in shape. This is most marked in the left foot. Both feet trip in walking, especially when the boy is tired. Both present the typical appearance of talipes equino-varus. The calf muscles are not enlarged, but feel firm. The tendo Achillis of both sides, of the left most markedly, is tense. The other muscles of the body seem natural. The organic reflexes are undisturbed. All the superficial reflexes are elicited with ease. The patellar tendon-reflexes and those of other tendons are exaggerated. Ankle-clonus is pronounced on both sides. Sensory functions are normal. The muscles of the left calf exhibit a diminished reaction both to faradic and galvanic stimulation. The muscles on the front of the same leg appear more excitable than is normal; and in the case of the tibialis anticus and long extensors of the toes, there is evidence of qualitative change. Bald patches, like those found on the father's, are present on the boy's head; and the dorsal aspect of the patient's toes are reddened, and present the appearance of

[1] The parents' observations are of considerable value, as long experience of disease had led them to watch the early development of symptoms with care.

chilblains. There is no cerebral disturbance. The spine is free of obvious deformity, and there is no tenderness or hyperæsthesia. The other systems are normal.

*Remarks.*—The cases are of interest as illustrating the occasional occurrence of different types of nervous disease in one family. That of G. P. (senior) is presumably one of gradually advancing spastic paralysis, probably excited by injury in a constitution predisposed to such affection through the previous ill-conditions of life. This view is supported by the history of the onset and the mode of advance,—the gradual increasing motor weakness, with rigidity of the lower extremities, and the comparative absence, from the beginning, of sensory disturbance. The exact significance of the sensory area, which apparently now exists at the lower part of the spinal column, is a question of some difficulty, but the presence of the area does not materially militate against the view that has been adopted. In addition to the spastic phenomena, his nervous system affords evidence of depreciation in the alopecic patches and other trophic disturbances. G. P. junior presents the early symptoms of a similar affection. The history and general characters, coupled with the exaggerated deep reflexes and ankle-clonus, are in favour of this view The evidence of qualitative change in a single group of muscles was slight, and is insufficient to warrant dubiety with regard to the diagnosis, being, of course, capable of more than one explanation in harmony with this. Curiously enough, he, too, has bald patches on his head, closely resembling those seen in the father. A. P., on the other hand, is suffering from pseudo-hypertrophic paralysis, of which two older brothers have already died. He has no alopecic patches. The facts, therefore, suggest a transmitted tendency to primary lateral sclerosis, while the family history illustrates the known hereditary character of pseudo-hypertrophic paralysis; the former apparently passing in the direct line from father to son, the latter, as usual, through the mother to the male children only (see history). A further interesting question in heredity arises, in view of the fact that, of all the children, only the youngest son—born after the father's spastic symptoms had begun to develop—became affected in this special way, and this, apparently, in the absence of a direct, exciting cause.

In connection with the case of pseudo-hypertrophic paralysis, there are one or two other points of interest. The increased heat of the skin over the affected muscles speaks in favour of the view, that the seat of disturbance is the vaso-motor system. This receives important confirmation in the presence of the

well-marked goitre, without any indication of cretinism. Another curious fact is the presence of distinct pigmentation of the forehead and temples and backs of hands in a boy, who for months has practically been confined to the couch, recalling Dr. Gower's[1] case of pseudo-hypertrophic paralysis, seen along with Sir Wm. Jenner, where the skin of the temples, posterior axillary fold, abdomen, back and thighs, was so pigmented as to warrant the diagnosis of Addison's disease. The last point is the tendency to incontinence of urine, which, during the last few weeks, has begun to manifest itself—a late symptom to which reference has been made by Dr. Byrom Bramwell.[2]

[1] Gower's 'Pseudo-hypertrophic Paralysis,' p. 19, case 24.

[2] 'Lancet,' Aug. 9, 1879.

# HYSTERO-CATALEPSY IN A MALE; ATTACKS SUSPENDED BY TESTICULAR PRESSURE.

BY ALLAN M'LANE-HAMILTON, M.D. (NEW YORK).

THE following case is of interest, not only by reason of its rarity, but because of the success that followed an interesting therapeutical experiment. To Dr. Robert Abbe, who kindly called me in consultation, I am indebted for the greater part of the following notes.

C. G. B., aged 35, married eight years. Father addicted to the excessive use of stimulants, and irascible. The patient is an intellectual man, and one of superior capacity. Mother of opposite type, a beautiful woman of quiet, even temperament, free from any trace of hysterical nature. Patient rather fat, and of one hundred and seventy pounds average weight. Health ordinarily excellent, though he has suffered from what was generally believed to be chronic peritonitis of several months' duration, eight months ago. He also had an attack five years ago, of pneumonia, with sequelæ, which one of his physicians thought was mild cerebro-spinal meningitis. Three years ago, while labouring under business reverses, he clandestinely began to take morphine—in eighth of a grain pills. The morphine habit grew rapidly until date, and he has never stopped it, and has taken at times as high as sixteen grains a day, but usually three grains in two doses, morning and night. His intellectual habit has been less bright during this time, though I have usually regarded him as lazy in mind, and from a boy disposed to exaggeration. His mother and senior brother (who is now in business with him) have for years been half credulous of spiritualism, but the patient has always scoffed at them. On the 16th of February last, he had a chill, which marked the beginning of pneumonia of a well-defined croupous type. He began convalescence after the tenth day, and was nursed by a female trained nurse, who resorted to massage quite frequently to quiet him from nervousness, supposed to be due to discontinuance of the morphine, which was unadvisable under the circumstances. He entirely gave up the drug rather abruptly during the fever, though some

frequent and considerable hypodermic injections were given for the relief of pain up to the crisis. On Monday, the 2nd of March, when convalescence was progressing, and he had sat up for two hours during the afternoon for the second time, he retired in a comfortable mood. The nurse had been dismissed, and he was alone with his wife. About nine in the evening he said he did not think he would sleep well. His wife tried to soothe him, but in a few minutes he began to show nervousness, and acted queerly. He began to kick off the bed-clothes, and act as if in a fit of petulance or temper; then turned over and beat the pillow violently, as if to vent his feelings. This was soon changed into a state of mental pre-occupation and moaning, quiet conversation in secret with imaginary people, exclamations of, "Oh, mother, mother!" as if she was seen in a dream. His fists were clenched and relaxed alternately; eyes rolled up imploringly, and apparently fixed on space. The head was occasionally buried back in pillow. Has general anæsthesia, though moaning, as if in a dream, "My head—my head; it aches so!" Pupils rather widely dilated, but reacting to candle-light rather sluggishly. He did not vary much from this state all night, except to remain quiescent for two or three consecutive hours, with apparent insensibility, and mostly complete unconsciousness. Towards morning the spell seemed to relax, and he half awoke, dazed, and declaring that the night had been a perfect blank, though sometimes he had seemed to answer questions intelligibly.

He drank milk freely, and seemed to have come to himself, but soon lapsed again into the queer unconsciousness. Many such relapses occurred during the day at intervals of half an hour, or at times two or three minutes only occurring.

They always began by a muscular fixation of the head backward on the pillow, eyes rolled upward a little, and lids open. (When I saw the case with Dr. Abbe, there was a slight tremor of the lids.) The ophthalmoscope showed a perfectly normal retina. The functions remained normal. Urine was passed in ordinary quantities, and the pulse never varied from 90, throughout the beginning, middle, or end of the attack, or the intervals. Temperature 99½, not altered.

I saw him about twenty-four hours after the commencement of the attacks, and while sitting by his side he complained of headache, and after shuddering slightly, the fixation of the head just alluded to began to be apparent. There was first rolling upwards of the eyeballs, with a slight tendency to convergence, suspended respiration, and afterwards some slight oscillation of the eyeballs, and audible respiration. The colour of the face was, if anything, rather pale, and the lips were somewhat livid. When I extended the upper or

lower extremities, they remained in the position in which I had placed them, and there was slight, almost inappreciable, *flexibilitas ceræ*. There was no relaxation within a reasonable time—one or two minutes. With this condition there was very decided analgesia, and a pin was thrust into the surface, and the hair pulled, without any expression of suffering. The patellar tendon-reflex was, if anything, slightly exaggerated, though no cremasteric reflex could be evoked. In a period of about five minutes, there was some appearance of volitional return, for he opened his eyes, moaned, and placed his hands upon his head, and appeared to suffer. After rousing him, he was able to indistinctly call attention to his distress, but almost immediately he again became rigid. I this time extended and slightly adducted his hands and forearms, placing the tips of his little fingers and thumbs in contact, so that a sort of arch was presented. This implied a very delicate muscular co-ordination, and in a conscious, non-cataleptic person would require considerable effort. The position was maintained, however, without so much as a tremor, for two minutes. I then, bearing in mind the efficacy of ovarian pressure in corresponding states in woman, suggested to Dr. Abbe that he should make firm pressure upon one testicle. This he did, and almost *immediately* the rigidity relaxed, and the arms dropped. Coincident with this there was a return to consciousness.

Dr. Abbe, who closely watched the case, says, "Testicular pressure broke the charm, and they (the attacks) never returned." On the following evening he declared he was going to be nervous and sleepless again, and his wife said he began to act in the same way. I found him at 10 P.M. nervous, but apparently trying to control himself, yet kicking first one leg, and then the other, under the bed-clothes. He would lie quiet for a moment, and then snort and turn over.

I ordered his wife and nurse, who had returned, out of the room, and systematically bullied him for two hours, when he gradually quieted down, though not much sleep came.

During the catalepsy, his constant complaint on waking was that his head pained as if "*bursting*," front and back.

He had told me regarding sexual functions that, while vigorous, he had not had, he believed, more than two emissions during the act of coitus during two years past. The third day before the display above described was marked by three seminal emissions, without provocation or erection; they made him feel weak.

Others had occurred during the weeks he was in bed convalescing, and occasionally followed massage. The nurse had habitually taken temperature per rectum, and had noticed

an over-sensitiveness of the perineum, which made him squirm, and, on giving enemata, made her desist.

After the night of scolding he rapidly changed, and not a nervous sign appeared again. He walked out on the fourth day after, and was well in a week.

This, so far as I know, is the first reported case of hystero-catalepsy in a male, though I believe that most of these cases of catalepsy met with among young subjects, of either sex, present an hysterical element.

# A CASE OF MULTIPLE SIMULTANEOUS CEREBRAL HÆMORRHAGES, CAUSING HEMIPLEGIA AND OCULO-PUPILLARY SYMPTOMS.

BY W. HALE WHITE, M.D.,

*Assistant Physician to Guy's Hospital.*

JOHN R. B., æt. 44. Admitted into Guy's Hospital, Nov. 5th, noon. Eighteen months ago he had weakness in the left arm and leg, which came on slowly without any preliminary unconsciousness. He recovered his strength in about eight months, power returning first in the leg. On the morning of admission, whilst clipping a horse, the patient fell down insensible; he was not convulsed, but appeared "as if in a sleep." Within an hour he was brought to the hospital. He was then unconscious, and had passed his urine and fæces under him.

*On Admission.*—Face, pale. He lies on his back with extended thighs and legs, breathing stertorously and apparently in profound coma. Temp. 97·6; extremities cold; P. 56, hard and full; R. 32.

*Nervous System.*—The pupils are seen to be minutely contracted when the closed eyelids are raised; they do not react to light. Deviation of both eyes to left. Conjunctiva œdematous; corneæ sensitive to touch. No deviation of head. Jaws firmly closed. During inspiration alæ nasi dilate; during expiration the right cheek bulges more than the left, and the naso-labial furrow is less marked on the right side. The right arm and leg appear to be paralysed; there is slight rigidity of both. The thumb of the right hand is bent over into the palm and the fingers closed over it; when it is forcibly disengaged, it slowly returns to the same position. The left arm is freely moved about, and is occasionally convulsed, especially in the fingers and the flexors of the forearm.

*Heart.*—Obviously considerably hypertrophied.

*Urinary System.*—Urine pale; sp. gr. 1015, and contains a considerable amount of albumen.

1.45 P.M.—P. 72. The left eye is still deviated to the left; but the right eye was directed forwards. Pupils pin-point contraction, insensitive. Jaw relaxed and fallen. The right arm had been seen to move once or twice, as if involuntarily.

2.15 P.M.—P. 60; T. 97. Deviation of both eyes outwards; pupils still contracted.

3.15 P.M.— R. irregular, 24.

3.30 P.M.—Pupils dilated, but unequal; the right being much more dilated than left. Deviation outwards on both sides. Both arms are flaccid and mostly motionless, but both move occasionally.

4 P.M.—P. 95, weak and fluttering; T. 96.8.

4.30 P.M.—Death.

*Post-mortem Examination.*—Brain: all the three cerebral and the basilar arteries were thick and sodden-looking, no calcareous plates, or miliary aneurisms. The convolutions were much flattened. The left cerebral hemisphere was torn up by a large hæmorrhage, which had filled the ventricle with blood clot. The chief seat of the lesion was the internal capsule, and this together with the lenticular nucleus, external capsule and claustrum were completely destroyed. The antero-posterior extent of the injury was from the level of the anterior extremity of the corpus striatum back to the top of the posterior cornu. Externally, just the innermost part of the grey matter of the island of Reil was affected. Internally, the caudate nucleus was bulged over by the hæmorrhage into the right ventricle; the optic thalamus was destroyed, and it was apparently at the anterior part of this body that the intraventricular rupture had taken place. The septum lucidum was torn through; the right ventricle was full of blood, also the third and fourth ventricles, for some of the blood had passed down the aqueduct of Sylvius, which was full of clot; some blood had also passed out of the great transverse fissure to the base of the brain, and the clot was pressing on the left 6th, 7th, 8th, and 9th nerves, at their point of exit from the brain; the 7th, 8th, and 9th, on the right, were also pressed upon by clot, but only slightly. On section through the pons, a small recent hæmorrhage, about the size of a millet seed, was found exactly in the centre. Immediately under the centre of the floor of the aqueduct of Sylvius was an elongated hæmorrhage, which began at the extreme upper part of the aqueduct and extended nearly to its lower extremity. This elongated clot was about the thickness of a rather thick probe: round it were many punctiform excavations. In the descending motor fibres of the pons on the right side was a small old hæmorrhage, about the size of a pea, of a brownish colour. The kidneys were granular; the heart much hypertrophied, it weighed 26½ oz. The testicles were fibroid, and there was an old hydatid cyst between the liver and the diaphragm.

The interest of this case speaks for itself. During life some difficulty was felt in accounting for the symptoms, and considering the condition of the pupils, those who first saw him placed the lesion in the pons; but it seems to me that the fact, that the hæmorrhage was sufficiently great to produce insensibility and considerable lowering of temperature, should have made them consider, that it was hardly likely that a hæmorrhage into the pons, sufficiently extensive to produce both these symptoms, should yet not affect the motor fibres going to both sides of the body. But the post-mortem examination explained these difficulties: for undoubtedly the first thing that happened was that a large hæmorrhage took place in the left cerebral hemisphere, and, bursting into the ventricles, caused, in addition to hemiplegia, insensibility and lowering of temperature; at the same time, the pontin hæmorrhage in the floor of the aqueduct of Sylvius caused the contraction of the pupils. The bleeding, probably, did not stop from either hæmorrhage during the patient's life, for the varying oculo-motor and pupillary symptoms can be explained easily by the pontin hæmorrhage affecting gradually different parts of the oculo-motor and pupillary centres in which it took place. The rigidity of the right arm and the occasional movements of both left and right are interesting also, as showing some irritation of the motor fibres. As when the patient was first seen there were no symptoms of the clot pressing on the left 6th, 7th, 8th, 9th nerves, it is probable that the blood did not reach them till later on, and perhaps did not clot till after death. The case has been recorded, because it is not very common to get, as in this case, three simultaneous hæmorrhages, viz. one ventricular and two pontin, although I have recently made a post-mortem examination in which severe meningeal hæmorrhage was associated with hæmorrhage into the pons. The possibility of more than one hæmorrhage occurring at the same time should always be borne in mind, if, as in this case, the grouping of symptoms is at all peculiar.

# Reviews and Notices of Books.

*A Text-book of Human Physiology, with Special Reference to Practical Medicine.* By Prof. L. LANDOIS, of Greifswald. Translated from the Fourth German Edition, with Additions, by WM. STIRLING, M.D., Sc.D., Reg. Prof. Insts. of Med., University of Aberdeen. 2 vols. 8vo., with nearly 500 Illustrations. London: Griffin & Co., 1885.

THE first volume of Professor Stirling's translation of Landois' 'Human Physiology' appeared last year, and has been already noticed in these pages ('BRAIN,' vii. p. 534). With the publication of the second volume we have the complete work before us. The general impression left in the minds of most people by a general survey of its pages must, we think, be favourable, and the impression will certainly be strengthened by a more minute study of their contents. These are full and detailed, without being redundant; well arranged under distinct headings, and well indexed, so that any desired subject can be readily found without undue turning over of pages. The Text-book was already a good one in its mother tongue, it is in every way a better one in its English dress, and its title "Landois and Stirling" is quite justified by the amount of new matter which has been introduced by the translator. We cannot, however, help regretting the absence of references, which are so useful and prominent a feature in, e.g. Beaunis' 'Physiology'; in a two-volume work such as the present, which is to be used by students of physiology as well as by students of medicine, the lack of references is a serious want. The labour of making these at all complete is, however, very great, and it would hardly be reasonable to expect to find them in a first edition; but we may hope to see the want supplied in a future edition of a book which is to take its place as a standard Text-book for advanced students. It is doubtless after due reflection, that Professor Stirling has added largely to the histological and anatomical departments by illustrations as well as by descriptions—additions which, however, have added to the bulk and price of the Text-book more than to its value as a compendium of modern physiology.

Of the 650 pages contained in the second volume, more than 450 are devoted to the Nervous System. Section X gives the Physiology of the Motor Apparatus; XI, General Physiology of the Nerves and Electro-Physiology; XII, Physiology of the Peripheral Nerves; XIII, Physiology of the Nerve-centres; XIV, the Sense-organs; and these, the most important chapters of

Physiology to the readers of this Journal, are also the most complete and satisfactory as regards treatment and arrangement. The instruments and methods used by physiologists in the investigation of the nervous system are very fully described, and the descriptions are made the more intelligible by diagrams and figures to which Dr. Stirling has made many valuable additions; the laboratory aspect of the subject is such as is useful and suggestive to those whose studies are chiefly clinical, while the clinical aspect is well kept in view, and cannot but be useful to those whose pursuits lie chiefly in the laboratory. It is further an advantage, that the exposition is encyclopædic rather than critical; the student who consults the work finds facts and statements to inform his own opinion rather than ready-made opinions. This feature is of no small value in a Text-book, and the only desideratum that remains is a reference to the original sources of the quoted facts and statements. As regards phraseology, we think that is a secondary matter in a Text-book like the present; it is, however, a relief not to meet with German-English in a translation from the German, and this is ample evidence of skilful interpretation that has cost much care. To the qualification of centres, as centres *for* sneezing, *for* coughing, *for* vomiting, &c., exception might perhaps be taken as implying meanings rather more material than in reality, but for these Prof. Landois is responsible, and they are consecrated in most Text-books. On the whole we may feel assured that "Landois and Stirling" will be a leading text-book in this country, as "Landois" is already in Germany, and that it will exercise great influence in the teaching and in the study of physiology in the medical curriculum.

A. Waller, M.D.

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*Recherches expérimentales sur le mécanisme de fonctionnement des Centres Psychomoteurs du Cerveau.* Par le Dr. J. M. L. Marique. 1 vol. large 8vo., pp. 140. Paris: Delahaye, 1885.

This little book is an aggregation Thesis presented to the University of Brussels. The first part is historical and critical; the second, experimental and critical. We need not say much here of the first, which contains a clear *exposé* of the various lines of argument adopted at different times and by different authors, in their attempts to prove or disprove the theory of cortical localisations. In the second, the author endeavours to establish the reflex "psycho-motor" mechanism of the cortex, partly by discussion, but chiefly by experimental methods, of which we shall endeavour to give a clear though succinct account. The point to be established is that the motor area is excited to action only by stimuli arriving to it from the cortical "sensorium commune," through

the fibres of associations which connect the different convolutions or lobes with one another.

Anatomical considerations show that it is possible, by means of rather superficial incisions, to isolate the sigmoid gyrus of the dog from the other cortical tracts of the brain, without injuring the peduncular and callosal fibres. For whereas the latter sink vertically into the white matter of the hemisphere, the fasciculi of association between the gyrus and the frontal parietal and temporal lobes, run near the surface and in a horizontal direction.

In order to avoid the disturbing effects of the anæmia of the motor gyrus which would necessarily attend division of the pia mater around it, the following method was adopted by Dr. Marique. A narrow blade is plunged to the depth of seven or eight millimetres at one of the angles of the gyrus, into the substance of the second frontal convolution, close to the motor area. The knife is then tilted, so that the point moves towards the median line, and a transverse incision is effected, dividing the grey matter and subjacent white matter as far as the corpus callosum, but leaving the pia mater and its vessels untouched. A second transverse incision is likewise made at the other angle of the gyrus; and a third, in a longitudinal direction, joins the inferior extremities of the two former.

In one series of experiments (ten dogs) the following plan was adopted. After the gyrus had been exposed, the animal was allowed to recover from the anæsthetic. Two hours afterwards it was etherised again, and the precise situation of the motor centres was determined by the induced current. The three incisions were then made as first described; and after the waking up of the dog, the effects produced were duly observed. Finally, a controlling experiment, to prove that the fibres connecting the cortex with the peduncle were intact, was made; the animal was etherised a third time and the induced current applied to the motor centres, when the characteristic movements on the opposite side of the body occurred.

The phenomena observed on the return of consciousness after the isolation of the motor tract by the three incisions are the same as those that follow the complete ablation of this tract. The animal first tries to bring into a natural position its extended limbs, but succeeds only in moving those on the same side as the lesion, and falls on trying to stand. After a short time it recovers the power of walking, but imperfectly, treading on the dorsal aspect of the carpus; and it cannot step over obstacles. Dogs accustomed to give their paws when ordered have lost the power of doing so with the paretic limb. The locomotor symptoms tend to disappear after the second day; but there remains for many weeks an inability to perform with the affected limbs action requiring a higher grade of co-ordination, or acquired by a process of training.

In a second series of experiments (five dogs) the three incisions were not made all at once, but singly, in order to determine the effects produced by each. The anterior transverse incision was

found to have but a very slight and transitory effect, nothing but a trace of paresis of the opposite limbs being observed. The inferior longitudinal incision produced a more pronounced weakness in those parts; whilst the posterior transverse cut was found to be almost as effectual as the complete isolation of the gyrus.

These results agree with what one would have expected, on the hypothesis of a reflex cortical mechanism. For, in the dog, the frontal region being but slightly developed as compared with the posterior convolutions, the share it takes in the psychomotor processes must be proportionally limited. The development of the temporo-sphenoidal region, on the other hand, places it in an intermediate position; and the experimental results, as we have seen, tally with the anatomical data.

Dr. Marique does not think that the symptoms of paralysis were in any way due to circulatory disturbances in the motor area, and he answers by anticipation this and other possible objections to the method he has employed. It is to be regretted, however, that he did not use the antiseptic method in his experiments. Still they appear to us sufficiently free from fallacies to establish his conclusions, which after all are but a confirmation of the view held by many, that the motor areas of the brain are to its sensorial tracts, what the anterior cornua are to the posterior in the spinal cord. We need not follow the author in his discussion of the question of substitution. He practically adopts the theory that in the dog the lower centres are able to carry out the simple co-ordinated movements which make up the bulk of this animal's motor manifestations. The proof that the other half of the brain does not take up the functions of the injured hemisphere lies in this fact—that destruction of the former does not paralyse the limbs, the control of which it is supposed to have assumed. It seems to us also much more rational to assume that the corpus striatum, which in man and the higher apes may be considered as a mere surviving organ in the process of obliteration, occupies in the dog and other animals of the same grade an intermediate position: the cortical centres are only beginning to assume some of the functions of the lower centres which still retain a considerable potential power. We must not hastily assume, however, that because a dog deprived of his cortical centres reacts to ordinary stimuli in an apparently normal manner, it has not suffered loss in the psychical sphere, and that "substitution" can ever be complete. At any rate, much more complete observations than those we possess are required to establish this point.

A. DE WAITEVILLE.

*Zehn Vorlesungen über den Bau der Nervösen Central-organe.* By Dr. LUDWIG EDINGER. (1 vol. 8vo., pp. 138, with 120 figs. Leipzig: F. C. W. Vogel, 1885.)

*Scheme of the Functions of the Cerebral Nerves.* By Professor JACOB HEIBERG, of Christiania. Wiesbaden: J. F. Bergmann, 1885.

THESE Lectures, delivered in 1884 to an audience of medical men, are distinguished chiefly by the numerous, and for the most part really admirable drawings of the various parts of the central nervous system, and illustrating both structure and naked-eye appearances. Many of the figures are diagrammatic, and if these are not always strictly accurate, they invariably express what they are meant to express, leaving no doubt on the subject. We do not know of similar detailed representations of cerebral structure in any single work. For the most part details are named on the figures; an immense economy of time, saving the annoyance of searching through a long letterpress description, to find perhaps that the structure sought is not named there after all, but in the text. It is not always possible thus to indicate structures on the figure itself, but there is no reason why it should not be oftener done than it is. The artistic work, in the great majority of these figures, is much superior to the woodcuts of our English textbooks.

The first Lecture is devoted to a short account of the history, and the methods of investigation, by which the structure of the central nervous system has been discovered. In other lectures there is no mention of authorities, and conflicting opinions are not as a rule stated. Lectures II. and III. give an excellent account of the gross anatomy of the brain, Dr. Edinger here as elsewhere making his figures serve in place of long word-descriptions. The remainder of the Lectures describe the structure and connections of the brain and spinal cord. Dr. Edinger explicitly states that he keeps as far as possible to received doctrines, without attempting to incorporate the results of his own observations on disputed points. Hence there is little ground for criticism. We need say, regarding the text, only that it is clear and uninvolved, and will be readily intelligible even to English readers. It should, we think, be specially useful to those who, wishing to take advantage of German work on the nervous system, are not always sure as to the identity of the structures named. In the text, Dr. Edinger gives both the Latin and the German nomenclature.

This is an ingenious attempt to represent, on two pages, the functions of the cerebral nerves. The method adopted is that of coloured type, motor nerves being indicated by red, sensory by yellowish-brown, and special nerves by blue. Compound nerves are indicated by the names being printed partly in each corresponding colour. The result is certainly striking, and conveys a large number of facts in small space. The student for whom it is

partly intended might, however, with the help of coloured pencils, make a synopsis which, although not so pretty, would be more useful to him than Professor Heiberg's ready-made one. The practitioner may with advantage use it as a reminder, but in doing so it will be well to remember, that there is more than a doubt as to the muscular supply of the palate muscles being derived from the facial, and also that the spinal accessory has other functions besides supplying the sterno-mastoid and trapezius muscles. The coloured printing and general execution of the synopsis is extremely good.

JAMES ANDERSON, M.D.

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*Report on Recent Advances in the Anatomy of the Nervous System.* By JAMES ANDERSON, M.D.

WHILE our knowledge of the motor tracts of the brain and cord is far from complete, it is fairly definite and extensive. The same cannot be said of our knowledge of the sensory tracts, due partly to the greater inherent difficulty of the inquiry, partly to what may be called the accident, that the sensory fibres in the medulla oblongata and pons lie deeper than the motor.

Dr. E. C. SPITZKA makes a considerable contribution on this head in a series of articles on the Anatomy of the *Lemniscus*, with remarks on centripetal conducting tracts in the brain (*New York Med. Record*, Oct. 11th, 1884, *et seq.*). It will be remembered that the lemniscus appears as a flat triangular field, lying on the lateral aspect of the isthmus cerebri, stretching between the superficial origin of the fourth nerve above to the crusta below, overlapped in front by the transverse fibres from the posterior quadrigeminal body, and overlapping behind the anterior crus of the cerebellum. This, however, is only the exposure of a great tract which extends from above the pyramidal decussation through the interolivary region to the thalamus, gradually becoming more and more remote from the median raphé, while another detachment remains near it.

Professor Spitzka remarks, that while centrifugal fibres run clear from cortex to spinal cord, centripetal fibres are everywhere interrupted by ganglionic masses; an anatomical fact corresponding with the related physiological fact, that while the motor impulse is a comparatively simple affair, the co-ordination, for example, of space impressions from skin and ear into a general space sense is a complicated affair, requiring the intervention most probably of the cerebellum.

He quotes an important case published by him. "An elderly gentleman, after a small hæmorrhage in the pons, which caused unilateral ataxia of the opposite extremities, lived six years, during which time an intense secondary degeneration developed in the *descending* direction, and terminated after decussating in the opposite nuclei of the columns of Goll and Burdach, which were likewise atrophic." The columns of Goll and Burdach, at least

in their upper parts, degenerate centripetally, that is, towards their nuclei, so that apparently the two segments of this tract "degenerate toward an interpolated ganglion, justifying the assumption that functional impressions converge toward it, though not excluding the possibility of a continuous transmission brainward of sensory impressions at the same time." It will at once be seen that Professor Spitzka is justified in terming this suggestion a "startling innovation." The degeneration in this case descended through the interolivary tract of Flechsig, crossed in what Dr. Spitzka terms the *piniform decussation* in front of the hypoglossal nuclei (Meynert's fine upper pyramidal decussation), and so reached the degenerated nuclei of Goll and Burdach. Brown-Séquard found, that in spinal hemisection the muscular sense does not follow the rule of spinal decussation, but suffers on the same side as the motor function. Dr. Spitzka's case, along with others quoted by him, explains this apparent anomaly. The piniform decussation consists of fibres passing from the lenticular nucleus by the ansa lenticularis (linsenkernschlinge) which goes to form the main part of the lemniscus, and also a tract of undecussated centripetal fibres conveying muscular sense which probably ascends to the capsule independently, and so to the parietal lobe in close relation with the motor tract, a fact explaining the frequent disturbance of muscular sense present in hemiplegia. This latter, the lemnisco-pedal tract, or Rinden-Schleife of Monakow, degenerates downward only; the remainder, which is the main part of the lemniscus (formed by the olivary fasciculus and fibres from the piniform decussation), degenerates both upward and downward in different parts.

While acknowledging that to the atrophy and degeneration methods of Flechsig we owe the great advance during the last ten years in the anatomy of the brain and cord, Dr. Spitzka considers that certain of his assumptions are unjustifiable. For example, in his classical case of absence of the cerebellum, the cerebellum is far too important an element in the central mechanism to be eliminated without producing far-reaching defects of development elsewhere. Again, he states that from his observations he is becoming more and more convinced, that absolute reliance should not be placed upon the apparent continuity of myelinic development.

Dr. ALLEN STARR'S Prize Essay on the Sensory Tract in the Central Nervous System (*Journal of Nervous and Mental Disease*, July 1884) contains not only an admirable summary of our present knowledge—and our present ignorance—on this subject, but also the detailed examination of the brain of a microcephalic infant, and much careful collation of cases. The infant lived for one week; it took nourishment, and cried like a normal infant, but made no voluntary movements, and seemed incapable of perceiving sensations. Medulla, pons, and cerebellum were present, but small. The prosencephalon was undeveloped, except a small detached mass between two layers of dura mater. The

thalamencephalon was represented by two grey masses lying anterior to the pons and cerebellum, from which arose the optic tracts and nerves, small in size. The other cranial nerves, from 3rd to 12th, were present. The pyramidal tracts were entirely absent. The interolivary tracts were partly developed, and showed the decussation of the fibres from the nuclei of Goll's and of Burdach's column. These fibres, the lemnisco-pedal tract of Spitzka, pass up in the external part of the lemniscus, and would appear, from their being present in this case, to develop from below upward, although, as we have just seen, Spitzka's case throws doubt upon this. The major part of the interolivary tract, *i.e.* the internal part of the lemniscus coming from the lenticular nucleus by the ansa lenticularis, was absent, developing apparently from above downward. Thus a continuous tract could be traced upward from the nuclei of Goll's and of Burdach's columns, across the median line through the interolivary tract into the lemniscus, and upward into the tegmentum of the crus cerebri, where it lay in the external lateral part of that body. The crustæ were entirely absent, as also the corpora quadrigemina. The red nucleus, and probably also the substantia nigra, were present. The formatio reticularis, the posterior longitudinal bundle, the nuclei in the floor of the fourth ventricle, the cerebellum and its peduncles, were normal. All the motor tracts, therefore, were absent; all the sensory tracts present. From this and two similar cases he concludes, that the sensory tracts must lie in the formatio reticularis in the lemniscus and interolivary tracts.

From this case and an analysis of other cases and observations, Dr. Starr draws the following general conclusions:—

1. In the cortex of the brain, sensations of touch, pain, temperature and the muscular sense are perceived.

2. These perceptions occur in the grey matter of the anterior and posterior central convolutions and of the parietal convolutions; sensations from one side of the body being perceived in the opposite half of the brain in a more intense degree than in the same half of the brain.

3. The various sensory areas for various parts of the body lie about and coincide to some extent with the various motor areas for similar parts—the area for the face, arm, and leg lying in the lower, middle, and upper thirds of the sensory-motor region respectively.

4. While the motor area is confined to the central convolutions, the sensory area includes, to some extent, the convolutions of the parietal lobe which lie adjacent and posterior to them. It is therefore more extensive than the motor area.

A.—The surface of the body is connected with a definite region of the surface of the brain by distinct tracts which convey sensory impressions. These impressions enter the spinal cord by the posterior nerve-roots, and then ascend in different tracts.

(*a*) The impressions destined to awaken the sensation of touch pass at once to the opposite half of the spinal cord, to a great

extent if not entirely, and ascend in the posterior white columns, the impressions from the legs passing in the post rior median, and those from the arms in the posterior lateral columns. On reaching the medulla these impressions pass to the formatio reticularis of the same side on which they were in the cord, and ascending in this tract through the pons and crus, reach the internal capsule, where they are conducted by a tract lying in the inner half of its posterior third to the corona radiata, whence they diverge to the cortex of the central and parietal regions, the impressions from the leg being perceived in the upper third of these regions, and those from the arm being perceived in the middle third of these regions.

(*b*) The impressions destined to awaken the sensations of pain and temperature also cross the median line immediately after entering the spinal cord, and pass up in the grey matter of the cord, probably in its posterior inner part. On reaching the medulla they enter the formatio reticularis, and from this point their course is identical with that of the impressions of touch already traced.

(*c*) The impressions destined to awaken the sensation of the location and of the motion of a limb ascend in the spinal cord, in the posterior white columns of the same side upon which they enter: the muscular sense from the legs passing up in the posterior median, and that from the arms in the posterior lateral column. On reaching the medulla, these impressions pass to the opposite side through the sensory decussation, then ascend in the interolivary tract to the pons, where they enter the lemniscus, and gradually turn outward from the median line as they pass up through the pons. In the crus, these impressions are conducted by the lateral lemniscus, which lies in the outer third of the tegmentum, and which passes directly into the internal capsule, there being situated in the middle part of the posterior third, external and in close approximation to the sensory tract of touch already described. From this point, the course of these kinds of impressions cannot be distinguished from one another, and their termination is in the cortex of the central and parietal regions, the muscular sense of the leg being perceived in the upper third, and that of the arm in the middle third, of those regions.

B.—Sensory impressions from the face enter the pons by the sensory root of the trigeminus, and pass downward to sensory cells which lie in the lateral portion of the formatio reticularis, and which are ranged in a column extending from the junction of the upper and middle third of the pons, to the lower limit of the medulla. The upper portion of this column receives the fibres from the upper branch of the nerve; the middle portion from the middle branch, and the lower portion from the lower branch. The course of sensory impulses, from these sensory cells to the brain, is in the lateral part of the formatio reticularis of the same side upon which they enter, up to the junction of the upper and middle thirds of the pons, where they cross the median line and join the sensory impressions from the body in the formatio reticularis. In the crus cerebri, these impressions pass in the outer half of the tegmentum, and, thence entering the posterior third of the internal capsule,

pass on to the corona radiata. In the divergence of fibres in the corona, these impressions pass along the lowest radiation, and thus reach the lower third of the central and parietal regions, where they are perceived. It is, as yet, impossible to distinguish between the course of tactile, painful, and muscular impressions from the face to the cortex of the brain.

C.—Sensory impressions from the great cavities of the body enter the spinal cord by the posterior nerve-roots and are probably received by sensory cells which lie in the inner and posterior part of the grey matter of the spinal cord—the Clarke column of cells. From these cells, these impressions pass outward to a white column lying in the lateral periphery of the cord—the direct cerebellar column—in which they pass upward to the medulla, and on through the restiform body to the cerebellum, to terminate either in the nucleus dentatus, or in the cortex, or in both. The existence of a decussation of these impulses is undetermined, but, if it occurs, it must take place soon after their entrance, as each direct cerebellar column passes to that hemisphere of the cerebellum of the same side upon which it lies. The impressions thus transmitted to the cerebellum are not those of touch, pain, temperature, or the muscular sense, and are probably impressions connected with the functions of vegetative life and destined to awaken reflex actions.

In a recent issue we discussed the first two of Professor Burt Wilder's "Cartwright Lectures on the Methods of Studying the Brain." The third Lecture (*New York Med. Journ.*, April 5th, 1884, *et seq.*) is divided into two parts; the first devoted to the completion, correction and general castigation of our anatomical textbooks, and chiefly of the two which Professor Wilder tersely denominates "Quain" and "Gray;" the second, to a discussion of the methods of illustration and description, with a revision of encephalic nomenclature. Professor Wilder considers that we place too implicit a trust in the two works alluded to, and treats of their omissions, inaccuracies, and ambiguities.

As to omissions, we may at once set at rest the anxieties of any junior readers by saying, that those pointed out by Dr. Wilder are neither very numerous nor very important. We scarcely think that the editor of 'Gray's Anatomy' would claim for it completeness in every detail. The omission from it, for example, of a description of the tractus transversus pedunculi of Gudden, rechristened the Cimbia by Dr. Wilder, can scarcely be said seriously to impair its value as a student's textbook of anatomy. The same omission even in 'Quain's Anatomy' cannot be regarded as a grievous sin, although doubtless it ought to have been mentioned, seeing that that work, at least in this part of it, is no longer a student's textbook, but a work of reference. The account of the nervous system in 'Quain's Anatomy' is well and copiously illustrated, but Dr. Wilder reproaches it for not containing diagrams of the bulbus cornu posterioris and of the cauda striati, both described in the text. Dr. Wilder considers that the striæ longitudiuales callosi are merely the prints of the anterior cerebral

arteries; but opinions may very well differ on this matter, and the absence of reference to this cannot be characterised as an omission. This completes the list, and its shortness is surely a strong testimony in favour of our textbooks. The inaccuracies, Dr. Wilder admits, are "comparatively trivial." He first shows elaborately that the centrum ovale majus does not exist, because the upper surface of the corpus callosum is concave transversely, and convex antero-posteriorly, so that a horizontal section on the level of the corpus callosum would open the lateral ventricles. This simply means, that the section to expose the broad white surface called centrum ovale majus is not a plane surface, but a doubly curved one. The complete circumscription of the end of the descending horn of the lateral ventricle is implied in the usual accounts, but is certainly not definitely stated as it ought to be. A more important correction, however, is contained in Dr. Wilder's description of the commissura fornicis, the thin lamina which unites the two thicker lateral masses of the fornix. This lamina is a secondary formation, the two halves of the fornix being originally distinct, a fundamental fact, the recognition of which is necessary to an intelligent conception of cerebral architecture.

For seven years, Dr. Wilder states, he has been in the habit of beginning the account of the brain, whether for anatomical or physiological purposes, by a description of the cavities, their mode of origin, their succession, &c., the parietes being described as built up around them, and gradually differentiated. There is much to recommend this method, and we fully agree with Dr. Wilder that, both in the figures and in the text of our standard anatomical works, there is much indefiniteness on the details of what he terms "cœlian circumscription," that is, as to the boundaries of the ventricular cavities. We cannot share Dr. Wilder's scepticism as to the existence of the Foramen of Magendie. Difficult as it is to believe in its existence *à priori*, there can be no doubt whatever of the fact, explain it as we may. To the Foramen of Monro Dr. Wilder does full justice, and to any one who really wishes to understand the part of the cerebrum usually known as the transverse fissure, the relation to this of the velum interpositum, and the precise formation of the Foramen of Monro, we recommend a careful perusal of Dr. Wilder's description. He insists also on the fact, now well recognised, that the "fifth ventricle" is no part of the true ventricular cavity, has in the adult no communication with it, and never had any, but is merely a part of the longitudinal fissure separated off by the downward curvature of the corpus callosum.

Dr. Wilder makes some excellent suggestions regarding figures which he would use not merely to supplement, but, as far as possible, to replace description. He would have them made more numerous and more definite, and he suggests rules for the placing of them in order to facilitate easy reference and comparison. Some of these rules we should have transcribed, but that Dr. Wilder, as already stated, writes in an unknown tongue, *e.g.* he tells us that "transections of a symmesal object should form a horizontal

syntropic series." The general object is to secure uniformity, and thereby save the labour involved in mentally reversing figures. One recommendation we should very gladly see generally adopted, namely, that the full technical names of parts should as far as possible be given on the figure, or by the side of it, with indicator lines. The economy of time by the general adoption of this plan would be very great. The last part of the third Lecture, devoted to a revision of nomenclature, was not delivered, but the substance of it is given in the *New York Med. Journ.*, March 21st and 28th, 1885, to which we must refer the reader.

## Abstracts of British and Foreign Journals.

**Fol on the Micrococcus of Hydrophobia.**—At the meeting of the Académie des Sciences held on the 14th of December, 1885, Professor H. Fol, of Geneva, stated that after many endeavours he has found a method to demonstrate in the spinal cord of hydrophobic animals, certain bodies which are not to be seen in the healthy tissues. The microscopic sections must not be thicker than ·02 of a millimetre, and the hardening and staining processes carried out according to the methods of Ehrlich and Weigert.

Under such conditions, and in specimens otherwise perfect, groups of rounded bodies—micrococci in all probability—are seen to be lodged either in the lamellæ of neuroglia, or sometimes in the annular spaces comprised between the cylinders (stained dark blue by the hæmatoxylin) and the sheath of Schwann (buff coloured). Occasionally they are found in certain cavities, the size of a myelin fibre, of which the histological nature is unknown.

The globular bodies are perfectly spherical, defined, and stained dark violet; they usually lie scattered, not in strings; though at times are found to form figures of 8, showing a scissiparous mode of multiplication. Their average diameter is ·0002 millimetre.

An appropriate medium, sown with rabid brain-matter, and kept at a certain temperature, shows a slight turbidity which on the fourth day falls to the bottom of the liquid. This deposit has the power of giving hydrophobia to animals inoculated with it, though the period of incubation is longer than when the original matter is used. The same deposit applied to a cover-glass, dried and treated with the bichromate and copper solution, then coloured and uncoloured in the same way as the cord, shows the same groups of micrococci, stained dark violet. Inoculation of cultures of more than six days old does not produce any marked symptoms. It would be interesting to ascertain whether this is due to an alteration of the virus, and whether animals so treated become proof to the poison.

**Horsley and Schäfer on the Muscular Contractions which are evoked by Excitation of the Motor Tract.**—At a meeting of the Royal Society, held on the 10th of December, 1885, the authors described the results of a large number of experiments undertaken, in order to determine the character of the muscular contractions which result from excitation of the several parts of the motor tract, especially with reference to the rhythm with which the skeletal muscles respond to such excitation.

For the purpose of their experiments they considered the motor tract under four heads, viz.:—1. Its commencement in the nerve-cells of the cerebral cortex. 2. The connection of these cells with the lower nerve-centres by the nerve-fibres in the corona radiata. 3. Its continuation along the medulla oblongata and medulla spinalis (including the nerve-cells of those structures). 4. Its peripheral continuation along the motor nerves.

Their method of proceeding was to excite these several parts in succession, and record the contractions of one of the limb muscles upon a moving blackened surface, either by directly connecting the tendon with the lever of a myograph, or by Marey's method of transmission by tambours and india-rubber tubing, the time being simultaneously recorded upon the moving surface by a clock marking seconds. Usually the rate and duration of the excitation were also recorded by a small electro-magnet. Besides the contractions resulting from electrical excitation, they frequently obtained an accidental record upon the moving surface of spontaneous or voluntary contractions of the muscle, the responses of which to electrical excitation of the cortex cerebri they were preparing to record, and they have thus been able to compare these records of voluntary contractions in animals both with the results of electrical excitation of the several parts of the motor tract in the same animals, and with records of voluntary contractions in the human subject. They also studied, in the same way, the epileptoid contractions which are often found to follow a period of electrical excitation of the cortex cerebri in animals, and compared these epileptoid contractions with numerous others recorded by one of them from cases of true epilepsy and other affections of the nervous system (in man and animals), accompanied by rhythmic muscular movements.

*Results.*—It has generally been admitted by authors, that so far as regards the rhythm of muscular response, the result of exciting either the cerebral cortex or any other part of the motor tract is precisely the same as that which is well known to be the case with

the excitation of the motor nerve, namely, that for all rates of excitation the rhythm of muscular response is identical with the rhythm of excitation. The experiments of Horsley and Schäfer show, on the contrary, that this statement only holds good for low rates of excitation up to about ten or twelve per second, but that for all higher rates of excitation of the cortex cerebri, corona radiata, or medulla spinalis, the muscular response does not vary with the rate of excitation, but maintains a constant rhythm, which is independent of the excitation rate and approximate to ten per second.

The muscle curves which obtained from different mammals as the result of successive excitation of the cortex cerebri, corona radiata after removal of the superjacent cortex, and of the cervical cord after section below the medulla oblongata, are very similar to one another, and exhibit along their course, both at the commencement and during the whole extent of contraction of the muscle, small but distinct undulations following one another at the rate of about ten per second, with very considerable regularity, although in a few instances the rhythm may be a little slower or faster than these (eight to thirteen per second are the extreme variations observed). These undulations have the same rhythm and character whatever the rate of excitation (unless this be allowed to fall below about ten per second). Moreover, precisely similar undulations are always visible upon the myographic curve of all voluntary or spontaneous contractions (including reflex contractions) both in the lower animals and in man.

It is further noted, that in the record of the contractions of epilepsy there can frequently be seen marked upon the larger curves, produced by the relatively slow clonic spasms, smaller undulations succeeding one another with a rhythm of eight or ten per second. In some cases the clonic contractions themselves may attain this rate, but they are then always simple and without any indications of smaller waves.

In a very few instances out of a very large number of experiments, there occurred upon the tracings obtained as the result of rapid excitation of the cortex cerebri, corona radiata, and medulla spinalis, besides the usual well-marked undulations of the rate of about ten per second, other very minute waves upon these undulations, corresponding in rhythm with the rate of excitation. These were the only occasions in which we have obtained results at all similar to those mentioned by Franck and Pitres.

The authors showed tracings indicating the general nature of

their results, from which they draw the following and main conclusions: 1. That the normal rate of discharge of nervous impulses from the motor nerve-cells of the spinal cord along the motor nerve-fibres is approximately 10 per second. (The rhythm of a clonus (*e.g.* ankle-clonus) is also about 8 or 10 per second, and that of strychnine tetanus in the frog, as indicated by the electrical variations of the muscle current, has also about the same rate (according to Lovén). 2. That in the case of nervous impulses reaching these nerve-cells in more rapid succession than about 10 per second, a process of summation occurs within those nerve-cells, so that the rate of discharge remains about the same in all cases. 3. That the nervous impulses which produce a voluntary contraction also traverse the motor nerve-fibres at about the same rate. There is, however, no evidence to show whether this rhythm of the volitional impulses is generated in the cells of the cerebral cortex, or in the cells of the lower nerve-centres. 4. That the slower rhythm which is often exhibited in epileptoid contractions is the result of a further summation, but there is no distinct evidence to show where this occurs. 5. That occasionally, though rarely, the summation of rapidly succeeding nervous impulses may be only incompletely effected within the nerve-cells of the spinal cord, or may not occur at all. In these cases results similar to those of Franck and Pitres are obtained.

**Hale White on the Pathological Histology of the Semilunar and Superior Cervical Sympathetic Ganglia** (*Trans. Roy. Med. Chirurg. Soc.* 1885).—This paper is founded on an examination of about a hundred and fifty sections of the above ganglia, from which it is concluded that the external appearances of the ganglia are of no importance whatever in pathology, unless they be obviously affected by malignant disease, abscess, tubercle, &c., or have become implicated in some inflammatory condition of surrounding parts. The typical ganglionic nerve-cell resembles other ganglion cells; it is larger and more rounded than those from the anterior cornua. No inference can be drawn from the number of nerve-cells to be seen in a section, for the normal arrangement is irregular, the sections are not in the same plane, and all the ganglia are not the same size. Many cells have no processes, and some have neither nucleus nor nucleolus. As a general rule, the cells are wasted in patients who have died of wasting diseases; but this is subject to many exceptions. Pigmentary degeneration may or may not be accompanied by a diminution

in the size of the cell, and is almost universal in the sympathetic ganglia; perhaps this may be the cause of some of the slight ailments from which we all suffer. In the present state of our knowledge, the varying appearances from pigmentation have no significance. Pigmentation of nerve fibres does not occur. The amount of fibrous tissue varies very much in different specimens, and in different parts of the same specimen. The superior cervical ganglion contains more blood-vessels than the semilunar; in the latter there is no constant arrangement. In chronic Bright's disease the small arteries are thickened; otherwise in this malady the specimens are normal. In acute inflammation of the ganglia the section is obscured by the enormous number of escaped white blood cells, there is also multiplication of the connective tissue-cells. This may be accompanied by congestion. It has been found only in diabetes and purpura hæmorrhagica, and is the only pathological lesion about which one can speak with certainty.

A. DE WATTEVILLE.

**Fournier on the Mental Disorders of the Præataxic Stage of Syphilitic Locomotor Ataxy** (*L'Encéphale*, Nov. 1884). Extract from a series of Lectures on the Præataxic Stage of Syphilitic Tabes.—Mental disorder, the Author states, may occur in various stages of Locomotor Ataxy.

(I.) It may, in some exceptional cases, mark the very commencement of the disease. In one patient, who eventually suffered from well-marked tabes, that disorder was ushered in by the following mental symptoms: dulness and blunting of the intellect; slowness of ideation, inaptitude for any occupation requiring close attention; rapid cerebral fatigue caused by the least work; in a word, intellectual torpor, intellectual asthenia. This condition lasted about a year, and then, singularly enough, entirely disappeared, apparently yielding to antisyphilitic treatment.

Another patient of this class, before becoming ataxic, became incapable of following his usual employment; without being incoherent he had curious bizarre ideas; he was sad, melancholy, depressed, and during several months was pursued by thoughts of suicide. After awhile all these symptoms passed away to be succeeded, however, by others of the usual tabetic character.

From the above it would appear, that in the præataxic stage of tabes we may meet with transient mental affections, just as at about the same time, certain forms of paralysis occur which are of equally short duration. Thus the paralysis of ocular muscles, so

common a forerunner of ataxy, may have as a pendant certain forms of paralysis or paresis, so to speak, of the intellect, equally destined to disappear with more or less rapidity. The author thinks that probably these two orders of symptoms have a similar origin.

Occasionally the mental disorder, appearing at the very commencement of the præataxic stage, may be permanent, the tabes being then inaugurated by symptoms resembling those of ordinary general paralysis, which the author has elsewhere described under the name of "syphilitic general pseudo-paralysis."

(II.) It is much more common, however, for these psychical phenomena to appear at a time when the præataxic stage has made some progress. That is to say, they do not usher in the disease, except in the rare cases mentioned above; but they may be the first symptoms of a cerebral character, being themselves followed by others, such as vertigo, congestive attacks, aphasia, convulsions, hemiplegia, &c.

Clinically in one group of cases the symptoms are as follows:—weakening of the memory, modification of character, of temper, of the habits; an intellectual asthenia slowly progressing towards a final condition of hebetude. Of all the intellectual faculties the memory is that which, in most cases, is soonest and most severely affected, and this peculiarity, it may be remarked, resembles what occurs in cerebral syphilis, in which disorders of memory are common, and frequently severe.

This enfeebling of the memory in most cases progresses slowly; but its onset may be sudden, occurring after a congestive attack, or, what is more usual, after an epileptic crisis. Sometimes it even appears to increase by sudden gradations, each epileptic seizure or congestive attack leaving an increased weakness of memory, and making a notable stage in the progress of the amnesia.

Finally, in the præataxic stage we may, in some rare cases, meet with transient attacks of amnesia lasting from twenty minutes to half an hour, the memory then remaining for a time uncertain and faulty, but gradually recovering.

In another group of cases, temper, character, daily habits, are modified; and this is what enquiry into such a case reveals:—the patient has undergone a sudden moral transformation; has become reserved, gloomy and morose; he then loses his spirits and his vivacity, caring for nothing—either work or pleasure. He becomes idle and indifferent, inert and apathetic, and even ceases to care for himself. Now and then suddenly his dulled vitality wakes in the form of impatience, anger, and motiveless passion.

Or, in a third class, the intelligence becomes diminished, just as in cerebral syphilis; there is a species of intellectual asthenia, a slowness of ideation; conception of ideas is less facile, less clear, and less precise; aptitude for mental work, for business, for every occupation requiring an association of ideas is diminished; brain-weariness is brought on, not only by real intense thought, but by ordinary reading, by prolonged conversation, or even by listening to a stage play. As time goes on, the mental disorder increases, and the patient becomes incoherent and truly insane, falling into a condition of incoherent hebetude, this being but a stage in a disease which ends in that abolition of the mental faculties called dementia. It is rare, however, for the mental degeneration to progress thus far before the appearance of the usual symptoms of motor inco-ordination.

Twice the author has seen Tabes ushered in by a series of cerebral symptoms, more complex, and almost identical with those of General Paralysis of the Insane, to which he gives the name of "Syphilitic pseudo-paralysis." The symptoms in cases of this kind are as follows:—(1) Loss of memory. (2) Stammering, hesitation, slowness in delivery of words, a sing-song speech, omission of syllables in words. (3) Mental disorders, such as dulness, slowness, and stupidity, followed by more marked signs of mental deterioration. The patient is said to be "wrong in the head." He is unfit for business, is forgetful, makes mistakes, and after a while enters into another phase. He then shows great hebetude, but has none of those delusions of grandeur so common in ordinary general paralysis; he is rather in a happy state of torpor, giving way frequently to motiveless laughter, all desire and all will being lost. Then come on motor troubles, clumsiness, and muscular weakness, with a slow, hesitating, uncertain gait, inequality of pupils, difficulty and hesitation in articulation, tremor of the lips and of the facial muscles when the patient tries to speak. After a while he becomes incapable of uttering a complete sentence, an idea being only expressed by a few words slowly and painfully uttered; but still a certain degree of consciousness and even of lucidity is preserved, and the patient manages to answer a few questions and execute a few voluntary movements. In a later stage the pupil is immobile; there are amblyopia and amaurosis, incontinence of urine, abolition of the reflexes, with undeniable ataxy, which is, however, less marked than in ordinary cases of tabes. After long vegetating in this miserable state, the patient enters the final stage of his malady, his mental faculties becoming

gradually extinct. At the autopsy we find general thickening of the membranes, and especially of the pia mater, with lesions of the grey substance of the convolutions and enormous dilatation of the ventricles. In the cord we find the lesions characteristic of tabes.

Here, the author thinks, we have either a cerebral syphilis with consecutive tabes, or tabes commencing in the cerebral form, the latter being, in his opinion, the more probable; and he considers the malady remarkable in that it assumes the form of general paralysis, or, to be more exact, the form to which he gives the name "pseudo-paralysis" of syphilis. He is thus of opinion, that syphilitic tabes may be initiated by a series of cerebral symptoms, closely resembling those of ordinary general paralysis.

**On Disorders of Sensation in Cerebral Hemiplegia.** By Dr. Legroux and Dr. De Brun (*L'Encéphale*, 1884).—The disorders of sensation met with in hemiplegia are here classified into five groups:—

(1.) Sensation is unaffected, the paralysis being purely motor.

(2.) The anæsthesia affects the whole surface of the paralysed members.

(3.) The anæsthesia is absolute in one extreme segment (thigh or foot, for example), whilst it is much slighter in the middle segment, and still less in the other extreme segment. Thus, the foot may be absolutely anæsthetic, the leg less so, and the thigh almost unaffected. These difficult degrees of sensibility are not sharply defined, but fade gradually from one into another.

(4.) The external half of a limb has its sensibility normal, or slightly enfeebled, whilst the internal half is almost absolutely anæsthetic.

(5.) The anæsthesia is in patches of irregular distinction, zones of absolute anæsthesia being surrounded by surface but slightly affected; or we may find patches on an anæsthetic limb where touch remains almost normal. In this variety the patches of altered sensibility have sharply d fined margins, and instead of gradually passing away, the anæsthetic zones may shift about from day to day; a patch yesterday anæsthetic may to-day be normal.

The explanation of this oscillation of the anæsthesia is difficult; but since the cerebral lesion does not shift, it is concluded that the sensory disorders of hemiplegia do not obey the same laws as the motor affections, and do not correspond to fixed localised lesions of

the cortex. These disorders of sensation present other particulars, in which they differ from the motor affections. With respect to the mode of onset, for instance, we may often see the motor paralysis increase for three or four days before arriving at its maximum of intensity, while the anæsthesia attains its height at the very outset; or the motor disturbance increases whilst the anæsthesia is diminishing.

Many writers have shown the possibility of cure in these cases of anæsthesia by methods such as metallotherapy, usually applied in the sensory affections of hysteria. The authors then show how, if small patches on these anæsthetic limbs be rapidly pricked with a needle, the sensation will return, and this even in cases of long-standing hemiplegia with contraction.

The anæsthesia of cerebral hemiplegia presents many points of resemblance to that of hysterical, syphilitic, or toxic origin, showing that it is due to some alteration in the blood, or to a sudden or gradual modification in the pressure in the cerebral vessels. The oscillations, fluctuations, and variability of the anæsthesia can only be explained by cerebral phenomena, which, though tangible enough, are certainly transient. This view is supported by a consideration of the sensory phenomena so often premonitory of hemiplegia due to atheromatous vessels, which, closely resembling those under consideration, is caused by an alteration in the cerebral circulation. Again, the anæsthesia seen at the outset of general paralysis, is due to a congestion of the cortex preceding the chronic encephalitis.

Thus it seems extremely probable, that the disorders of sensation in question are due, in many cases at least, to an alteration of the cerebral blood-pressure. Of course it will be understood, that those cases in which the anæsthesia is caused by a lesion situate in the posterior part of the external capsule are here excluded from consideration.

**On the Slaty Discoloration of the Brain in General Paralysis and its connection with Bedsores.** By Dr. Baillarger (*Annales*, 1884).—This paper treats of a discoloration of the brain met with in hues varying from slate to almost black, and sometimes described by the term greenish, most commonly observed at the autopsies of general paralytics who have suffered from bedsores.

As a rule, the author finds this discoloration confined entirely to the cortical grey matter, being sharply defined by the junction

of the cortical with the white substance; but now and then it is observed in the basal ganglia.

The tissue thus affected is generally, but not invariably, softer than the surrounding convolutions, and is at times in a state of ramollissement. The base of the brain and the cerebellum are the parts most commonly thus affected, but the surface of the hemispheres does not always escape. The microscope reveals a granular stroma, with scattered nerve filaments and numerous cholesterine crystals. According to Calmeil, the discoloration is due to the infiltration of the affected tissue by numerous pus-corpuscles.

A fœtid odour is noted in about half the cases, but may be absent even when the softening is well marked.

The pia mater and arachnoid may present only the changes of general paralysis; but more commonly there is a purulent infiltration of the pia, at times completely hiding the convolutions. The arachnoid cavity may be empty, but generally contains a varying quantity of flaky serosity, a fœtid odour being occasionally present. Changes similar to these are met with in the cord and its membranes, with perhaps even greater frequency than in the brain.

These appearances are most commonly found at the autopsies of general paralytics, presenting large and deep sacral bedsores, and are due in many cases to an ascending cerebro-spinal meningitis; but it is important to notice that the discoloration in question has been met with when the meningitis was absent.

The author divides the cases into two classes:—

(1.) Those in which we find cerebro-spinal meningitis with infiltration of the pia mater with an ichorous pus. Here the discoloration is probably due to a simple imbibition of pus by the subjacent grey matter.

(2). Those in which the discoloration is observed without the meningitis. In such cases the explanation above given will not hold, but the change is attributed to an inflammation of the affected tissues, due to the deposit in them of ichorous pus absorbed from the bedsore by the blood-vessels, and characterised especially by the putrid odour and tendency to gangrenous degeneration.

That we most commonly find this lesion in the brains of general paralytics is readily explained by the relative frequency of bedsores in the later stages of that affection.

Now and then a purulent spinal meningitis is set up by a bedsore, without being accompanied by any discoloration of the brain.

R. Atkinson, F.R.C.S.

**Westphal on the Patellar Reflex in Diagnosis.** WESTPHAL (*Archiv f. Psych.* Bd. xv. p. 731) was consulted in 1878 by a man aged 45, who had exhibited secondary symptoms of syphilis four years before. He was hypochondriacal, and extremely nervous and anxious about himself, and had recently had a transient attack of hemianopsia ("he saw the half of things"), and amnesia. There was an absence of all symptoms of nervous disease, with the solitary exception, that the patellar reflex could not be evoked. This, however, was proof to Westphal of an affection of the external parts of the posterior columns of the cord in its upper lumbar portion, or at the junction of the lumbar and dorsal portions. Two years later there was commencing optic atrophy in both eyes, which in another two years had progressed to absolute blindness. And then, suddenly, an attack of acute mania with grandiose ideas, and slight impairment of speech developed, and the patient died. During the whole course of the case there were no symptoms of ataxia, or indeed of any affection of gait, and there were no noteworthy pains; there was, however, at times, some difficulty in emptying the bladder. The *post-mortem* confirmed the diagnosis. There was sclerosis of the posterior columns throughout the cord, the disease being most extensive in the lower part of the dorsal division of the cord. Westphal discusses the etiology of these cases. He thinks that the syphilitic taint is only of secondary importance; the prime factor is an inborn psychical tendency, a peculiar disposition of the elements of the brain and spinal cord.

**Remak on the Tendon-Reflexes and the Reaction of Degeneration.** REMAK (*Archiv f. Psych.* Bd. xvi. p. 240) has studied the relationship that exists between the tendon-reflexes and the reaction of degeneration, with a view to aiding the differential diagnosis of amyotrophic paralysis of spinal, and peripheral origin, respectively. He summarises his results in the following conclusions:—(1) Exaggeration of the tendon-reflexes (more especially the phenomenon of ankle-clonus), with partial reaction of degeneration, has only been observed in cases of amyotrophic lateral sclerosis. (2) A normal condition of the tendon-reflexes with well-marked partial reaction of degeneration has only been observed in atrophic spinal paralysis, poliomyelitis anterior. (3) Absence of the tendon-reflex occurs, (*a*) with abolished excitability of the nerves, in all severe cases of amyotrophic flaccid paralysis, whether of spinal (poliomyelitic) or peripheral (neuritic) origin—and where there is resolution, the tendon-reflexes are still absent long after the

galvano-muscular reaction of degeneration has disappeared; (*b*) in the lighter cases of primary peripheral degenerative neuritis of mixed nerves, in which perhaps there is no paralysis; (*c*) in cases of absolute peripheral paralysis (e.g. from compression), even though there is no reaction of degeneration.

**Sakaky on Degeneration of Peripheral Nerves in Tabes Dorsalis.** WESTPHAL, seven years ago, examined the cutaneous nerves of the lower extremities in a case of tabes, and discovered that the number of double-contoured nerve-fibres was greatly lower than in normal nerves. Déjérine, three years ago, observed similar changes in two cases, which he described as peripheral neuritis, and regarded as a parenchymatous affection. The same author has recently reported two cases which presented the symptoms of tabes, but which showed no disease of the spinal cord, though the cutaneous nerves were atrophied. Sakaky (*Archiv f. Psych.* Bd. xv. p. 584) gives the result of a very careful examination he made of the nerves in a case of tabes. His observations show that in tabes there may be atrophy of the peripheral nerves, especially of the cutaneous branches. He is unable to state where the atrophy begins; in some nerves the peripheral portion was diseased, in others the central portion. He thinks that the presence of anæsthesia may perhaps be an indication of the atrophy of a nerve.

**Kirchhoff on the Localisation of the Ano-vesical Centre.** A man, aged 30 (*Archiv f. Psych.* Bd. xv. p. 607), fell from his horse, and fractured the first lumbar vertebra. Paralysis of the lower limbs, retention of urine, and loss of power over the sphincter ani at once appeared. The paralysis of the lower limbs gradually diminished, and in less than a year had disappeared. The retention of urine gave way in three weeks to incontinence, which, with the paresis of the sphincter ani, lasted until death. The patient died of pyelonephritis, a year and a half after the accident. The *post-mortem* showed lesion of the conus terminalis at the place of origin of the third and fourth sacral nerves. This is the situation of the ano-vesical centre in man, and it corresponds with the position of Stilling's sacral nucleus.

**Zacher on Spastic Symptoms in General Paralysis.**—ZACHER (*Archiv f. Psych.* Bd. xv. p. 359) concludes his paper on General Paralysis, by some observations on the spastic symptoms that sometimes occur during the course of that disease.

He finds that the spastic symptoms of general paralysis differ in important respects from the spastic spinal paralysis of Erb and Charcot. In the initial stage of the former there is a peculiar awkwardness and slowness in all the movements, that seems due to a mental want, to the absence of the proper ideas of the required muscular movements, rather than to the muscular stiffness and tension, which operate in spastic spinal disease. Then, again, there are remarkable variations from time to time in the state of the tendon-reflexes, the muscular tension, and the muscular contractures, which are in striking contrast with the regular progression and steady advance of the symptoms in spastic spinal paralysis. In general paralysis the contractures occur almost contemporaneously in lower and upper extremities, and are in both instances flexures; whereas in spastic spinal paralysis, the lower limbs are generally first affected, and exhibit not flexures, but, if we may be allowed the term, extensures (*Streckcontracturen*).

What is the pathology of these spastic symptoms? In two of the cases there was a primary systematic affection of the pyramidal strands quite independent of the cerebral disease; in another case there was a primary non-systematic affection of the cord, which implicated the pyramidal strands, but was found elsewhere and partook of the nature of a multiple sclerosis. Are the spastic symptoms due to lesion of these spinal pyramidal strands, and to lesion of these alone? Zacher answers in the negative. For, there may be spastic symptoms in general paralysis without any disease of the cord; and in the cases reported in this paper there was seen to be a close correspondence between the spastic symptoms and certain brain symptoms, e.g. the paralytic attacks that occurred periodically, and which were probably due to affection of the motor zone of the cerebrum. Zacher concludes that the spastic spinal symptoms of general paralysis may be caused by pathological changes, either in the motor region of the cerebrum, or in the pyramidal strands of the cord, and he thinks that the peculiar course of the symptoms in general paralysis points to lesion of the cerebrum as the predominant factor in these cases.

Westphal states that spastic contractures and rigidity of the muscles never appear in the lower limbs, in cases of combined fascicular disease of the posterior and lateral columns, if the lesion implicates the root-zones of the lumbar portion of the cord. Zacher extends this rule, and enunciates it thus:—In combined disease of the pyramidal strands and posterior columns, spastic

symptoms will be absent in those parts of the body where the corresponding root-zones of the posterior columns are diseased.

The question arises, How is this prohibitive action of the posterior columns to be explained? It is plainly not in harmony with the theory, that spastic symptoms are due simply to direct irritation of the motor fibres of the pyramidal strands. Zacher thinks that the spastic symptoms must be attributed to a pathological exaggeration of certain normal reflex actions, which normal reflex actions may be exaggerated or diminished by influences acting through the pyramidal strands, either in their spinal or encephalic course.

**Thomsen and Oppenheim on Sensory Anæsthesia.**—The authors contribute a long article (*Archiv f. Psych.* Bd. xv. p. 559 and p. 633) on the occurrence of sensory anæsthesia in diseases of the central nervous system. The term is used to embrace anæsthesias of the special senses, and hemianæsthesias. The paper is illustrated by fifty-two cases, and can only be very briefly summarised here.

The sensory anæsthesias form a typical group of symptoms, the most constant member of which is the bilateral concentric limitation of the visual field; the remaining special senses and the skin and mucous membranes are frequently affected, but by no means so regularly as the visual field. True hemianæsthesias are rare; indeed an absolute hemianæsthesia, in the sense that the other side of the body is quite unaffected, is unknown. Sensory anæsthesia is either a permanent condition, and then there are generally striking variations in its intensity and area of distribution from time to time; or more rarely it is a transient condition. It is important to observe that in almost every case, psychical symptoms are present; there may be disturbances of consciousness—hallucinations, dreamlike states, &c.; or disorders of the affective faculties—irritability, depression of spirits, feelings of apprehension, &c. The degree of anæsthesia runs parallel, as a rule, with the degree of psychical impairment.

Sensory anæsthesia (including hemianæsthesia) is not pathognomonic of hysteria and hystero-epilepsy, as many are apt to suppose. It is found in epilepsy, alcoholism, neurasthenia, chorea; in certain states characterised by continuous nervous apprehension with restlessness; in cases of railway-spine and head-injuries, in multiple sclerosis, in organic brain-diseases, and as a sequela in various psychoses. The authors criticise Charcot's description of

hysterical hemianæsthesia, and dissent from several of his conclusions. They aver that sensory anæsthesia occurs in three forms: first, in conjunction with certain psychical symptoms, just mentioned, as a separate disease; second, in association with other functional neuroses and psychoses; and third, in association with gross organic lesion of the central nervous system. In this last case, it is not the result of any specially localised lesion, but of some general cerebral disturbance. The symptoms are accordingly of little value, as yet, in diagnosis or prognosis.

**Bernhardt on Nerve-stretching in Facial Spasm.**—BERNHARDT (*Archiv f. Psych.* Bd. xv. p. 777) reports the case of a patient who, after suffering four-and-a-half years from right-sided facial spasm, underwent the operation of nerve-stretching. The spasm disappeared, but a corresponding facial paralysis took its place. Four months afterwards the involuntary convulsive twitching reappeared. Bernhardt gives an analysis of the results of nerve-stretching in seventeen cases of facial spasm, and he comes to the conclusion, that the operation procures temporary relief only; but that it is worth performing, even to secure this temporary respite.

W. J. DODDS, M.D., D.Sc.

**On Spinal Sclerosis of Vascular Origin.** DEMANGE (*Revue de Médecine*, October 10, 1884).—Female, age 75. No history of syphilis. Pain in loins and lower abdomen; frequent micturition and catarrh of bladder. Later, pain in legs, spasmodic flexions of legs. Later still, permanent contractures, flexion of knees, adduction of thighs; some atrophy; absence of tendon-reflexes; spasmodic action of bladder. Slight systolic cardiac murmur. Finally, sores on the heels, rigidity of neck and muscles of back; death from bedsore.

*Post-mortem.*—In the heart, granulo-fatty myocarditis, and slight chronic endocarditis; atheroma of the coronary arteries. Atheroma of the aorta and the cerebral vessels.

The spinal changes are characterised by the author as interstitial myelitis, in distribution diffuse and perivascular. There was *posterior sclerosis* both in the columns of Goll and of Burdach, but sometimes of each separately, sometimes together: the sclerosis was not bilaterally symmetrical; sometimes the whole column was involved, somet mes it was entirely sound.

The *antero-lateral columns* were deeply affected, but the crossed pyramidal tracts were not accurately marked out; the direct

pyramidal tracts were for the most part free: the sclerosis extended round the cord to the anterior root-zones.

The *grey matter*: there was one patch of softening in the lower dorsal region; elsewhere it was intact, the anterior cornual cells were normal.

As to the minute anatomy—In the small vessels the endothelium was proliferating and thickened, the vascular walls were thickened and infiltrated with nuclei; the lymphatic sheaths of the vessels were filled with leucocytes. The large vessels were affected in a similar way. The sclerosis appeared to start from the neighbourhood of the vessels: in patches of a star-shaped form with ill-defined borders. The neuroglia was thickened; there were numerous spider-cells; the myeline sheaths were atrophied; the axis-cylinders were present, but not swollen.

The author considers the case to be neither combined systematic sclerosis, nor lateral amyotrophic sclerosis, nor disseminated sclerosis; but diffuse myelitis depending on general atheroma of the vessels.

**Acute painful Paraplegia.** DUMOLARD (de Vizille) (*Revue de Médecine*, July 1884, p. 532).—An affection observed by the author at Vizille. Six cases are detailed, and the general summary of the symptoms is as follows: The disease begins with lumbar pain and s iffness in the back; the pain may be acute from the onset, or may gradually increase in intensity. It sometimes passes up to the neck. Next, pain in the thighs and legs, not following the course of the sciatic nor limited to the joints, but involving the whole limb. The gait is limping. In severe cases there is slight febrile action, but as a rule the general health remains good, except for pain and sleeplessness.

In a few days the patient is confined to bed with the pain, which is very severe. The reflex function of the cord is much increased, so that the least communicated movement gives rise to a species of tetanus in the lower limbs. The patient is unable to use his legs and has difficulty in passing urine, though the author does not think there is any true paralysis. In rare cases the symptoms are abruptly transferred from the lower to the upper extremity.

The acute period lasts 10 or 15 days; the pain first disappears, then control over the limbs and bladder is re-established. In 30 or 40 days the patient is well.

The pains are not of a shooting character, but rather of the nature of muscular cramp and twitching. They are more severe

than the pains of myelitis, and there is no girdle pain. The cause is as yet unknown. The author compares the disease to an epidemic observed fifteen years ago, at Arannon, in Spain.

**On Tabetic Entorrhœa.** G. H. Roger (*Revue de Médecine*, July 1884, p. 555).—The occurrence of diarrhœa in tabes has been commented upon by various authors, *e.g.* Charcot, Vulpian, Fournier, Pitres, Putnam. Out of 32 tabetic patients examined by the author, 14 had diarrhœa; and in 5 of these cases there was reason to think it had special connection with the disease.

"Tabetic diarrhœa" may occur either as an accompaniment of abdominal pains (crises entéralgiques); or without pain (crises interrhoïques), as a mere intestinal flux. Generally it is an early symptom, sometimes occurring even before the lightning-pains, sometimes it dates from the same time as they do, and it may have a distinct relation to them.

The attacks of diarrhœa are sudden both in onset and disappearance, and no cause can be assigned for them. They may last from two or three days up to a week or more. The stools are from three to eight in number per diem, a watery or mucous liquid, in colour grey, greenish, or yellowish. The patient may be subject to these attacks for many years. Not unfrequently they are associated with gastric crises, sometimes with secretory disturbances, such as sweating, sialorrhœa, or polyuria. In a case given by the author the diarrhœa was associated with excessive flow of saliva, and both were checked by the administration of atropine.

**Left Inferior Facial Paralysis. Left Hemiplegic Epilepsy. Doubtful Diagnosis before and after Death.** Landouzy and Sireday (*Revue de Médecine*, December 1884, p. 984).—An unmarried female, age 29, whose personal and family history was unexceptionable, came to the Charité for the following complaints: left facial paralysis, involving the lower facial district only, of six or seven months' duration; attacks of convulsions involving the left side of the face, which had begun about the same time as the paralysis as mere twitchings, but had increased in intensity, and at the time of the observation amounted to epileptiform attacks, spreading to the left side of the neck and to the left arm. There was evidence of a slight mitral stenosis, but no other disease.

An attack soon after admission is described as follows:—Slight aura = sense of weight in frontal region, dizziness, tremor of facial

muscles; then sudden attack, stiffness and convulsive movements of left arm, rotation of head and eyes to left, convulsions of facial muscles, tongue protruded and torn by teeth; only slight tremor of left leg. The convulsions were purely unilateral. Duration half a minute to a minute. Consciousness not lost. Paralysis of the affected muscles, and chiefly of the face followed the attack. Maximum interval two or three hours; attacks sometimes subintrant.

The patient became worse, the attacks more frequent and followed by coma, the pupils unequal, the temperature raised. Vigorous treatment with iodide of potassium was instituted, but the patient died.

No lesion was found post-mortem sufficient to account for the symptoms. It is mentioned, however, that on the posterior part of the 2nd frontal convolution, and the corresponding part of the 3rd frontal (right side), there was a patch the size of a five-franc piece, of a pale pink colour. There was a similar appearance on the lower part of the right parietal lobule at the level of the pli courbe.

These appearances, the author thinks, were more likely the result than the cause of the convulsions.

The diagnosis made during life was "Jacksonian epilepsy," *i.e.* epilepsy symptomatic of an irritative lesion of a part of the right fronto-parietal lobe. The absence of an appreciable organic change post-mortem made this diagnosis doubtful; but there are also difficulties in the way of the other hypotheses which might be advanced, viz. (1) simple epilepsy, with convulsions of limited area, and without loss of consciousness; (2) hysterical attacks having a fatal issue.

**Case of Arsenical Paralysis.** By CHARLES K. MILLS, M.D. (*Journal of Nervous and Mental Disease*, January 1883).—The poisoning was produced by eating a pumpkin pie containing arsenious acid. The patient was a man aged 24, previously healthy. Vomiting and prostration were the only immediate symptoms. Six days after the poisoning, numbness and aching about the knees came on, followed by weakness in the legs. Three days later, the same phenomena in the wrists and fingers; then complete paralysis below the knees. A month after the poisoning, severe pain, beginning in the knees and spreading downwards, aching, boring, and lancinating in character; the hands being similarly but less severely affected; numbness of the legs also became extreme. The result of an examination two and a half months after the poisoning

was as follows:—Much wasting of the limbs. Legs quite paralysed below the knees: no definite contractures, but occasional cramping pains with involuntary flexion at the knees. Impairment of tactile sense, with extreme sensitiveness of muscles, up to the middle of the thighs. Farado-contractility of muscles absent in legs, diminished in thighs. With galvanism, contraction to medium currents, alteration of polar reactions. Upper limbs:—well marked, though incomplete paralysis below the elbows, affecting specially the supinators and extensors. Movements of thumbs and fingers impaired; well-marked contractures at elbows; Farado-contractility diminished; to galvanism, reaction of degeneration, though less marked than in legs. Hyperæsthesia of fingers. Transverse white bands were noted on the fingers.

Patellar-tendon-reflexes absent. Cremasteric reflexes vigorous, and presenting some abnormalities.

Bowels torpid, some dribbling of urine. No albuminuria. Pulse between 107 and 148. Temperature normal or nearly. The patient improved under treatment, and in three months' time seemed on the road to recovery.

Of six other persons who partook of the pie, four had symptoms of paralysis.

**McNutt on Double Infantile Spastic Hemiplegia** (*American Journal of Medical Science*, Jan. 1885).—Dr. S. J. McNutt reports a case of double infantile spastic hemiplegia, with notes of the post-mortem appearances, illustrated with seven cuts exhibiting the lesions found. This is believed to be the third, or, at the most, the fourth case of its kind upon record. Yet these cases do not appear to be so very uncommon, since four others presenting similar symptoms are known to be now in New York city. As a distinct condition, even simple infantile spastic hemiplegia has but lately received attention in text-books. For this reason, and on account of the difficulty of obtaining any comprehensive information on the subject, the collection of facts and theories presented in Dr. McNutt's paper is of value, and must lead to a further study of this interesting condition. Dr. McNutt has collected and tabulated 34 cases in which autopsies have been made, and each of them presented atrophy of the cerebral cortex, near the fissure of Rolando.

The subjects of infantile spastic hemiplegia may live to old age. The inception of the disease, however, always dates back to early childhood, or to intra-uterine life. At whatever age seen, its victims are characterised by more or less complete hemiplegic

motor inability, atrophy, and contractures, with or without aphasia, monosyllable utterance, dysphagia, dyspnœa, and idiocy, the latter being especially characteristic of the double affection.

The etiology of infantile spastic hemiplegia has been defined as primitive defect, arrest, encephalitis, and hæmorrhage.

Clinically the cases may be divided into three classes; those in which the inception of the condition precedes birth, those in which it occurs after birth, and those of which parturition is the cause. The paper concludes with a careful study of the differential diagnosis and treatment.

**Osler on Jacksonian Epilepsy and the Situation of the Leg Centre** (*American Journal of Medical Science*, Jan. 1885).—The author describes a case of Jacksonian epilepsy, the main points of difference between which and true epilepsy are, the slow onset, local in character, beginning in, or in mild attacks confined to, one limb or a single group of muscles; the gradual extension until the side is involved, or, in severe attacks, the entire body; loss of consciousness late, not early and sudden, as in true epilepsy; and, lastly, the muscular contractions are clonic.

His case lasted over fourteen years, the convulsions beginning in the left hand, at first monobrachial, then extending to the leg, afterwards becoming unilateral, and finally general; at first without loss of consciousness. For the first nine years of the illness, there were remarkable intermissions, lasting for six or seven months, once an entire year. Six years after the onset, the left leg got weak and stiff. For four years, the tenth, eleventh, twelfth, and thirteenth of the illness, the seizures were frequent. During this period, there were six weeks of unconsciousness in which the spasms were very frequent, fifty to eighty in the day. Ten months prior to the final attacks, there was freedom from convulsions. The intellectual faculties were unimpaired.

The case is unusual in the limitation of the lesion to the ascending frontal convolution and to its fasciculus of white matter, scarcely involving the grey substance which is commonly affected in cortical epilepsy. The accurate localisation and the remarkable absence of tissue changes in the immediate vicinity give the case the nature of an exact physiological experiment. With this limited lesion of the motor area there were permanent paralysis, with contracture of one extremity, and epileptiform convulsions. Another feature of interest in the case is the light it throws on the situation of the leg centre. The fibrous mass was situated entirely

within the anterior part of the paracentral lobule limited in extent, confined chiefly to the medullary fibres of the superior frontal fasciculus, and only touched the grey matter in places. A point to be referred to is the absence of the paralysis of the leg for the first six years, for, if the convulsions and monoplegia were caused by the same lesion, how explain the late onset of the latter? From the fibroid state of the tumour it might reasonably be inferred that it was originally larger and had shrunk, but the absence of puckering on the surface, and the way in which the margins merged with the contiguous parts, make it probable that the growth was always small; so small, in fact, that at one period of its development it may have caused sufficient irritation to induce the convulsions, and yet at the same time not involve the special fasciculi of white fibres to the extent of producing weakness of the leg, or monoplegia.

**Kemper on a Case of Lodgment of a Breech-Pin in the Brain; Recovery** (*American Journal of Medical Science*, Jan. 1885).—A lad received a compound fracture of the frontal bone, immediately above the right frontal sinus, by a bursting gun. The breech-pin was found imbedded in the brain, at a distance of half an inch, and was withdrawn by the aid of dressing-forceps. No untoward symptoms were developed until the evening of the fourth day, when a convulsion ensued because of pent-up pus, and after the removal of the cause no further trouble followed. The lesson to be derived from the study of the case is the necessity of maintaining free draining in cases of a similar nature.

**Eliot on Poliomyelitis Anterior in Adults** (*American Journal of Medical Science*, Jan. 1885).—The author describes a case of poliomyelitis anterior occurring in an adult. The progressive development of muscular weakness, unattended by febrile symptoms, but accompanied by diminution of the size of the limbs, by abolition of the patellar tendon reflex, and by sensations of numbness, yet without loss of tactile sensation, and without interference with the function of either the rectum or bladder, rendered the diagnosis clear and indisputable.

A large proportion of the reports of cases which have been published contain little or no information concerning the details of treatment, and in many others the multiplicity of drugs prescribed renders any reliable conclusions in regard to the effect of each almost impossible. Bromide of potassium, belladonna,

strychnia, ergot, and iodide of potassium have been most often employed, and most praised. Counter-irritation, baths, rubbing and exercise, and electricity are also included as important elements in most plans of treatment. From a careful study of the results of various plans of treatment as reported by various observers, Dr. Eliot deduces the following conclusions:—

*First.* Counter-irritation and ergot should be employed early in every case. *Second.* Massage and electricity should be used as soon as there is evidence of improvement. *Third.* Little, if any, effect can be expeeted from iodide of potassium. *Fourth.* Belladonna and the bromides should be used only with extreme caution. *Fifth.* Strychnia should be entirely avoided.

**Reichert on the Regeneration of the Vagus and Hypoglossal Nerves** (*American Journal of Medical Science*, Jan. 1885).—Since the time of Fontana the subject of the regeneration of cut nerves has been one of great interest and importance, and some experimental work has been done with more or less success. At the present time there seems to be no difference in opinion as to the fact, that fibres of the cut ends of nerves will unite with similar fibres; and that the regenerated sensory nerves will still convey sensory impulses and the regenerated motor nerve motor impulses. In the case, however, of the regeneration of sensory with motor fibres, there yet exists considerable uncertainty.

Dr. E. T. Reichert records some experiments which were made to learn if the fibres of nerves of entirely different origin and function would unite, and if regeneration should occur, to know the form of the return of function, or, in other words, to know if a motor nerve was capable of conveying impulses peculiar to another motor nerve. The vagus and hypoglossal were selected as being nerves of distinct origin and function, and in case of regeneration would probably afford the best facilities for accurate observation.

The experiments were performed on dogs, and it was found that the motor fibres of the vagus in all of the five dogs operated upon had actually become united to similar fibres in the trunk of the hypoglossal, and that the hypoglossal fibres conveyed impulses which were peculiar to the vagus apparatus. Moreover, that in at least one dog (the others not being examined in this way) irritation of the sensory fibres in the hypoglossal trunk gave rise to impulses which were conveyed by the sensory fibres of the vagus to the vagus centres, and produced effects like those induced by

excitation of the vagus trunk, thus showing in both instances that a motor or sensory nerve can convey impulses peculiar to another motor or sensory nerve of entirely different origin and function; and indicating that at least in some nerves the effects produced by impulses from the periphery are not dependent upon any peculiarity of impulses due to physiological peculiarities of the peripheral sense-organs or nerves through which the impulses are conducted, but upon the peculiar physiological properties of the nerve centres; hence we have respiratory movements, &c., occurring in the tongue brought about by impulses from the vagus centres through the hypoglossal nerve, and effects on the respiration, pulse pressure, and vomiting centre, through impressions carried to the vagus centres by impulses generated in the hypoglossal.

Not only did Dr. Reichert find motor fibres of distinct origin and function united, but we find among the vagus fibres at least three physiologically distinct sets of motor fibres united with fibres of the hypoglossal, viz. fibres conveying *inspiratory* impulses, fibres conveying *expiratory* impulses, and fibres conveying *œsophageal* impulses; the first two sets, no doubt, consisting of fibres of the vagus going through the recurrent laryngeal to the muscles of the larynx, and the latter set forming part at least of the fibres belonging in the same branch.

Another interesting fact to be noted is, that the sensory fibres in the trunk of the hypoglossal, at the point of union with the vagus in these experiments, are recurrent fibres (sensory fibres coming from the superior cervical nerves through the descending branch of the hypoglossal and running from the branch towards the centre), and accordingly conduct impressions normally, not directly toward the centres, as is commonly the case with sensory nerves, but first peripherally making a circuit, as it were, before reaching the centres; therefore, since the sensory fibres in the hypoglossal which united with the sensory fibres in the vagus, conducted impressions to the vagus fibres, it is obvious that these impressions were conducted in a direction opposite to that of the normal, thus offering corroborative testimony to the very interesting experiment of Paul Bert, in showing that sensory fibres can convey impressions in both directions.

**Cortical Lesions of the Brain.**—By M. Allen Starr (*American Journal of Medical Sciences*).—The author gives an analysis of 50 cases of cerebral disease recorded in America, selecting only

those in which there was a single lesion, of moderate extent, of considerable duration, and determined with sufficient accuracy post-mortem. A consideration of the symptoms, both positive and negative, in these cases leads him to the following results.

*Lesions of the frontal lobes* (excluding the ascending frontal convolutions)—Twenty-three cases of various kinds—wounds, abscesses, tumours. Decided mental disturbance occurred in one-half the cases; a disturbance which may be described as a loss of self-control, and consequent change of character; a loss of power to fix the attention, or to recognise the import of an act in connection with other acts. No symptoms of aphasia, no motor or sensory paralysis; blindness, when it occurred, was referrible to optic neuritis, and disturbance in the sense of smell to pressure on the olfactory bulb.

*Lesions of temporal lobes* (11 cases).—Such lesions may exist without any localising symptoms; but they may cause disturbances of the special senses of hearing and of smell; of hearing, probably, when the first temporo-sphenoidal convolution is damaged; of smell, when the inner sphenoidal convolutions are damaged. It must be remembered, however, that the cases bearing on the localisation of the sense of smell are few and that hearing may be impaired by pressure on the auditory nerve. In one case the supra-marginal convolution was affected also, and there was amnesic aphasia.

*Lesions of occipital lobes* (10 cases).—In these the most prominent symptom was disturbance of vision. (The author has already fully treated of the function of the occipital lobes with respect to sight. *American Journal of Medical Science*, Jan. 1884, abstracted in BRAIN,' No. 28.) In three cases there were mental symptoms—loss of memory, occasional maniacal excitement, inability to recognise friends. Lesion of the angular gyrus was found in a case of paraphasia with agraphia; the patient at one time had lost memory of the appearance of words.

*Lesions of the parietal lobules* (4 cases).—No symptoms could be with certainty connected with the lesion.

The author will, in a future paper, consider lesions of the central convolutions. The present paper indicates, by the method of exclusions, that the centres for motion and for general sensation must lie here.

**Multiple Neuritis.** By S. G. Webber, M.D. (*Archives of Medicine*, August 1884).

Case I.—Male, æt. 22. Well till a year ago; since then, attacks of darting pains in abdomen and back. For the last five days pain and swelling in legs and arms, headache and feverishness. On admission, tenderness over left radial nerve, biceps cubiti of both sides, and muscles of right forearm: over peroneal and sciatic nerves of both sides, over calves and thighs. Swelling of hands, feet, and face. Arms contracted, in position of partial flexion. Lower limbs more or less paralysed throughout. Plantar reflex most marked on left side; patellar reflex absent on both sides. Faradic contractility feeble in tibialis anticus, peronei, and vastus externus of both sides; absent in rectus femoris and vastus internus; in interossei, moderately good; in extensors of forearms, supinator longus, and biceps almost absent; somewhat better in triceps, deltoid and flexors; in facial muscles normal. Temperature raised for first three weeks (once 104·5°).

He was treated with salicylic acid, iodide of potassium and electricity. Could get about in 2½ months; and in 4 months was quite well. The patellar reflex had, however, returned only in the right leg, and that slightly.

Causes alleged by patient, that he had eaten some unwholesome meat, that he had "caught cold."

Case II.—Female, æt. 28. Well till two months ago, when she caught cold while menstruating. After this, pain in abdomen, followed by numbness and anæsthesia, with a (transitory) girdle sensation. Then the numbness left the abdomen, and passed to the knees and legs. Sometimes sharp pains, sometimes dead aching, in the legs. Unable to stand, can move legs and feet, but keeps them slightly flexed; passive extension of them causes disagreeable feeling at heart. Slight numbness of hands. Tenderness over muscles and nerve-trunks of lower limbs. Impaired sensibility in left leg; plantar reflexes good; patellar reflexes absent. Reaction to faradism normal in tibiales antici; poor in right peronei, gastrocnemius, and quadriceps cruris; poor in left gastrocnemius, absent in left rectus femoris. Subsequently the reaction was absent from both peronei and from left vastus internus. Galvanic reactions (both sides) normal in tibialis anticus; in peroneus, "slow but strong" to 12 cells; in gastrocnemius, "very sluggish" to 16 cells.

Pain and tenderness increased in legs, and spread to upper-

limbs; subsequently the legs became free; but the fingers anæsthetic and tender.

Treatment: salicylic acid, blisters, morphia, and compresses of carbolic-acid solution.

In five months could walk with assistance, the heels not touching ground. In six months she was well.

Case III.—Female, æt. 34. History of rheumatism and alcoholism. Attack of vomiting three months before present illness. Present illness three weeks' duration; began with pain and numbness in the legs. Now pain chiefly in joints and back; cannot bear finger ends to be touched; perspirations, delirium. Tenderness of muscles and nerve-trunks of limbs. Paralysis of hands, feet, and legs, and (?) thighs. Anæsthesia in left lower limb. Absence of plantar and patellar reflexes. Complains of choking. Death the day after admission.

Case IV.—Female, age not given. History of gonorrhœa three years previously, and of chronic alcoholism. Drinking bout four weeks before admission, followed by vomiting, delirium, and hallucinations. For some days had had aching pains in lower limbs, and numbness in hands and forearms. Severe pain on admission; knees flexed, pain on attempt to straighten them; loss of power in extensors of wrists and hands; tenderness over nerve-trunks and muscles of all the limbs. Sore throat, but diphtheria could be excluded.

The paralysis advanced; anæsthesia of fingers and feet appeared; patellar reflexes were absent. The voice became reduced to a whisper. She died seventeen days after admission, with paralysis of the diaphragm, having been too ill to admit of a detailed examination.

*Post-mortem.*—There were found small suppurating spots in the lungs, and suppuration in the pelvis of the kidneys; also disease of the liver. The brain and spinal cord were both healthy. The principal nerve-trunks of the limbs were found to be diseased; the disease being most marked in the lower end of the sciatic system; less marked, though still extensive, in the distal ends of the nerves of the arms. The changes were very similar to those found in secondary degeneration after section of a nerve, viz. breaking up of the myeline, which was reduced to fine debris, or even absorbed; destruction of the axis cylinders or solution of their continuity; presence of small masses (nuclei?) which stained deeply in carmine. These changes were much more marked at the distal than at the proximal parts of the nerves. Where least marked,

the change consisted only in an alteration of the myeline near Ranvier's constrictions, without increase of nuclei, and without break in the axis cylinder.

The author has observed no less than eighteen cases of this disease, and gives the following summary of his observations:—Ages of patients: minimum, 9 years old; maximum, 51; usually between 20 and 30; no particular preference for either sex; onset a week to four months before admission, recovery usually in a few months; death three times. Sensory disturbances usually prominent; tingling and numbness, then pain and hyperæsthesia, tenderness over muscles and nerve-trunks (especially the latter). Motor disturbances, at first stiffness due to pain, then paralysis; contraction of lower limbs in position of semiflexion is common. Electrical reaction of muscles altered; wasting of muscles and general loss of flesh; sometimes glossy skin, œdema of limbs, excessive sweating. Tendon-reflexes usually absent; skin-reflexes may be normal. Some fever at first; sometimes mild delirium or hysteria. Disease usually begins in legs; may spread to arms, or even respiratory muscles. Diagnosis from anterior poliomyelitis, progressive muscular atrophy, lead-palsy; or in certain cases from rheumatic fever or spinal meningitis.

**A Case of Acute Poliomyelitis in the Adult.** By JOHN VAN DUYN, M.D. **A unique Case of Poliomyelitis Anterior Acuta of the Adult.** By AMBROSE L. RANNEY, M.D. (*Archives of Medicine*, August, 1884.)—In both cases the authors seek to establish, that excessive muscular action of a limb has an influence in determining the part on which the disease chiefly falls. In Van Duyn's case, the patient had been engaged a fortnight previously in "putting the stone," using principally the right arm. The motor paralysis (which was accompanied by a soreness and tenderness extending gradually over the whole body) began in the right hand, and spread in three days' time to all four limbs, sparing, however, the left forearm and hand; and when seen by the author about four months after the onset, the right upper limb, the left shoulder-muscles, and the left tibialis anticus were principally affected. In four months more, the left shoulder and tibialis anticus had recovered, while there was still much atrophy in the right upper limb.

In Ranney's case, the patient, a sawyer, was accustomed, in the course of his work, to work a treadle with his left foot, and push a board against a circular saw with his right hand. The paralysis

(which developed suddenly after sleeping on wet grass) was confined to the left foot and leg and right upper limb. The paralysis of the leg disappeared in a few weeks; but the right hand and arm remained, after nearly two years, paralysed and atrophied.

**Illustrations of the Anomalous Course of Posterior Spinal Sclerosis.**—By E. C. Seguin, M.D. (*Archives de Médecine*, October 1884).—In the first two cases the onset of the disease was marked by symptoms suggesting focal lesion of the lower part of the cord.

Case I.—Old lateral curvature of spine; syphilis fourteen years ago. Two years before he first came under observation, numbness appeared in left foot and leg and spread recently to right. Symptoms on first observation: progressive weakness of legs, slow micturition, diminution of sexual power; anæsthesia of feet: loss of patellar and plantar reflexes. No pains, no ataxia. A year later, ataxia had developed; and in another year or two, fulgurating pains, first in rectum, then in thighs and legs.

It is possible that a syphilitic myelitis occurred first, centrally situated, in the lower part of the cord, and posterior sclerosis followed secondarily.

Case II.—The patient, who had been previously healthy, except for a chancre some years before, came under observation for what appeared to be acute double sciatica, continuous pain in both lower limbs, which had come on suddenly after a chill. Numbness, slight loss of power, and slight emaciation of the legs followed; there was paralysis of bladder, absence of tendon-reflexes, well-marked ataxia of legs, griping pain in left chest. All the original symptoms disappeared, excepting the ataxia of the legs; the patient became subject to the ordinary fulgurating pains, and acquired the characteristic gait of locomotor ataxy.

The possibility of a local syphilitic lesion is again pointed out.

Case III.—Diplopia and atrophy of the optic nerves appeared four years before the pains set in.

Case IV.—The neuralgic pains had existed for twenty-nine years without ataxia of the legs. The patellar-tendon reflexes were absent. The patient died from cardiac and renal disease: post-mortem, sclerosis of the lateral parts of the posterior columns was found to exist.

Case V.—Characteristic pains for twelve years; slight anæsthesia of feet, absence of tendon-reflexes: no ataxia. Chronic arthritis of both knee-joints.

Case VI.—A patient had previously had two attacks of "cerebral congestion," and had once been "almost insane." He consulted the author for lightning pains of six years' duration. Absence of patellar tendon-reflexes, *slight* staggering with eyes closed. In three years time, typical general paralysis with grand delusions had developed.

Such a complication is well known to occur in the later stages of tabes: the author thinks it is unusual in the early (præ-ataxic) stage. In the present case, the cerebral and spinal affections may have been independent of each other.

**The Neurotic Disturbances after Joint Affections.** By Dr. George W. Jacoby (*Journal of Nervous and Mental Disease*, April 1884).—The paper is based on the observation of thirty cases; three of which are briefly detailed. These nervous disturbances may affect (1) sensation—producing hyperæsthesia or hyperalgesia (usually at an early stage), or anæsthesia (usually later), or neuralgic pains; (2) motion—producing paralysis of the muscles; (3) nutrition—producing muscular atrophy, hypertrophy of the subcutaneous tissue, sclerosis of the skin. The author's remarks chiefly relate to the muscular atrophy and paralysis. The course of the affection is as follows. At a variable time after an injury to a joint, which may have been severe or slight, the muscles become flabby, or even wasted, and lose their contractility. Paralysis (i.e. loss of volitional contractility) usually precedes this wasting; but on testing the wasted muscle it is found that their electrical contractility is affected also. Contractility both to faradic and galvanic current is much diminished, but there is no reaction of degeneration. This paralysis and atrophy affects, first and principally, the extensor muscles of the injured joint; but it may spread to others; and has been reported to pursue an ascending course and involve the entire half of the body (Révellont). In the case of the ankle- or wrist-joints, however, the extensors are not specially singled out, and the affection has a descending course. In the case of the finger-joints the interossei suffer most. The author believes that in paralysis of this kind there is always muscular atrophy, but that it may be obscured by hypertrophy of the subcutaneous tissue. In one of his cases, after injury to the knee, paralysis of the tri eps cruris was noted as soon as three days subsequently, and atrophy in four days more. The diagnosis from progressive muscular atrophy may be in some cases difficult, especially where the interessei are involved; the points of distinction are—that the paralysis precedes

the atrophy, disturbances of sensation are oftener present, the reaction of degeneration is absent. The prognosis is favourable: for treatment, the author relies greatly upon massage.

**Case of Nodular Tumour of the Corpus Callosum.** By FRANCIS A. MCGUIRE, M.D. (*American Journal of Neurology and Psychiatry*, May, 1884.)—This tumour (which appears to have been gummatous in nature) was a firm, irregular body, seated on the anterior edge of the corpus callosum, and covering about 2½ cm. of its upper face, also encroaching slightly on the curve towards the rostrum. Length 3½ cm., greatest diameter 2 cm. It consisted of a main mass continuous with the corpus callosum, with buds sprouting from it. The tissue of the corpus callosum was unsoftened, but the adjacent parts of the gyrus fornicatus on both sides was excavated; and also the cortex in the neighbourhood of the ascending and horizontal parts of the calloso-marginal fissure. There was also much thickening of the ependyma of the fourth ventricle.

The author does not connect the clinical phenomenon of the case with the tumour; the course of the disease had been briefly as follows:—syphilis in 1873, meningitis (?) in 1880, fair recovery; left facial palsy in 1882. Next year, headache, convulsive attacks, partial dementia; finally, other fits, in one of which he died.

**Case of Tumour in the Optic Thalamus.** By T. A. MCBRIDE, M.D. (*American Journal of Neurology and Psychiatry*, May 1884).—This tumour (also probably gummatous), of the size of a walnut, sprang from the optic thalamus at the junction of its posterior and middle thirds, and spread inwards into the ventricle, and through the corpus callosum into the longitudinal sulcus. [A transverse vertical section of the brain, made at the fissure of Rolando, thus shows the corpus callosum destroyed in the middle line, and to the right of the middle line.] The patient (who was deaf, incoherent, and dull) had left hemiplegia; and died with rise of temperature and delirium. He had complained previously of headache, and had been unable to walk, or to button his clothes, &c., so well as formerly.

J. A. ORMEROD, M.D.

END OF VOLUME VIII.

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TO

# BRAIN:

## A JOURNAL OF NEUROLOGY.

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